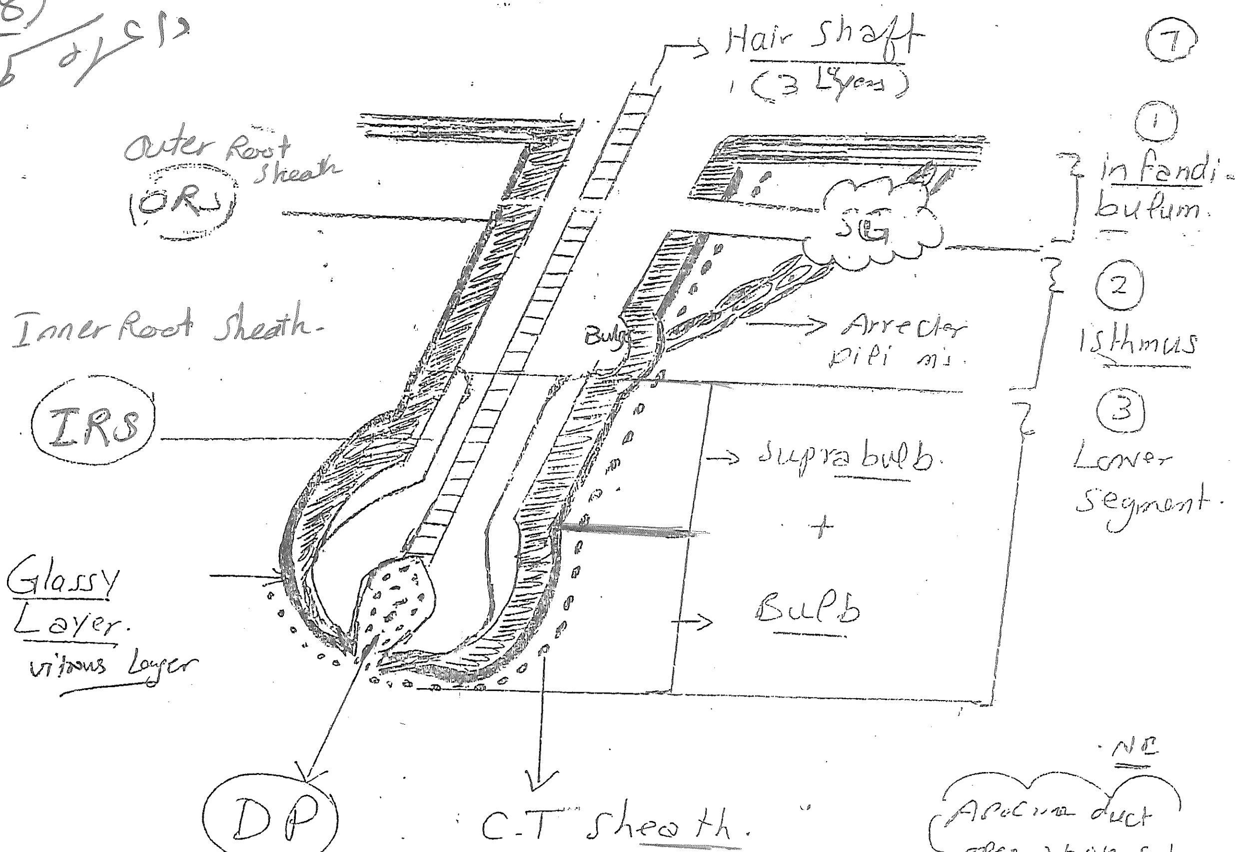
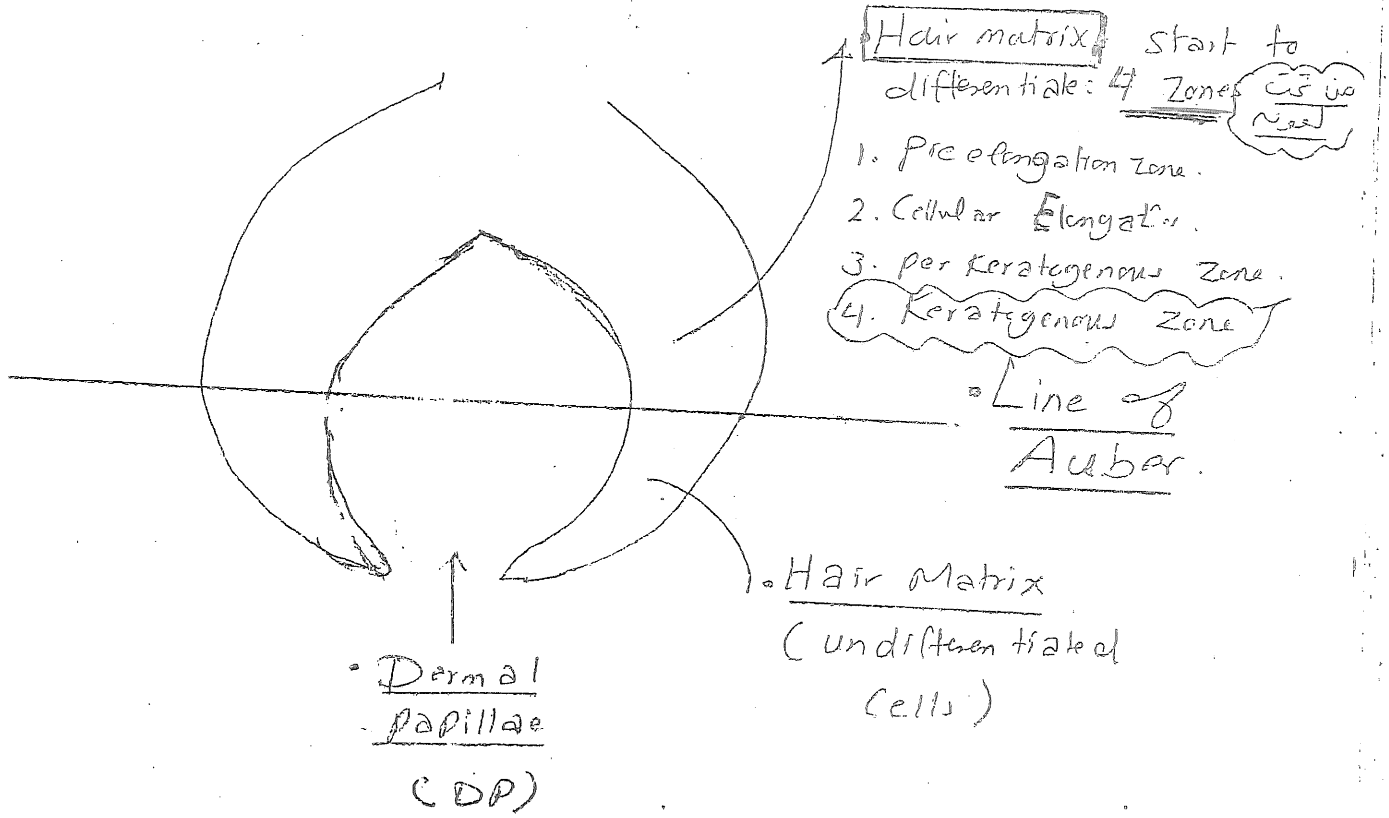
بسم الله الرحمن الرحيم

(Hair) (8) (7)



• NB
Apocrine duct open above Seb. duct in Apocrine sites.



Types of Hair shaft

① Lanugo

Lanugo

- Fine
- Soft
- unpigmented
- unmedullated

* Covers the fetus prenatally → sheds at birth.

② Vellous

2. Vellous

- Fine
- soft
- lightly pigm.
- unmedullated
- Covers most of body of Teenagers

< 1cm

③ Terminal

③ Terminal

- Coarse
- long >1cm
- Medullated
- pigmented
- Covers scalp, eyebrow, axilla, pubis & beard.

>1cm

④ intermediate

④ intermediate

- medullated & Moderate Pigmented.

(< Terminal)

≃ 1 Cm.

- According to the length :-
  - Vellous < 1 cm
  - Terminal > 1 cm
  - intermediate ≃ 1 cm.

- At puberty : Vellous hair of ← Beard, moustach, axilla, pubis — under effect of Androgen → Terminal hair.

Hair Cycle

- Hair doesn't grow continously (as do finger nails) but each follicle unsynchronized with the other follicles; undergoes cyclic Rythm of growth & rest phases.

- ④ Stages of Hair Cycles (A. C. T K)

Scalp Hair

• Anagen (active) phase	≃	3 ys	(I-VI) (90% of follicles)
• Catagen (involuting) "	≃	3 Ws	(< 1%)
• Telogen (Resting) "	≃	3 ms	(10% of follicles)
• Kenogen (Lag) "			

Kenogen (Lag) phase: is the (Lag phase) bet falling of Telogen & growth of New Anagen

وقع Telogen ومستنية اد anagen طلع ← فاضية (Empty follicle).

May occur: Normally or under effect of Androgen (Androgenetic Alopecia; as Androgen ↑ this phase).

• Exogen stage: (Shedding phase) (مرحلة سيمر ابعد Anagen): Represent the moment of shedding of Telogen & onset of New Anagen (Anagen IV).

Lash, Trunk, extrem.
• Anagen: 1-6 ms.
• Telogen: 2-4 ms.

Hair growth rate

✓ Scalp: 0.44 mm/d. (1cm/m. 15cm/y) (عشان مش تنسي ≃ ١ سم / شهر)
• Temple: 0.39 mm/d
• Body & beard: 0.27 mm/d.
short anagen hair → 5-6 months

Function of the Hair: [No vital funct.].

1. protection of < Scalp: from sundamage & Heat loss in cold climates. / Eye: from sun damage & droplets of sweat.
2. ↓ friction in intertrigenous areas.
3. May participate in Cut. sensory system (all follicles are ass. ē some sensory Nerves).
4. Cosmetic.

Alopecia = Hair loss

Hair diseases

1. Hair loss (Alopecia)
2. Hair Excess
3. Hair shaft defects.
4. Others.

- Cicatricial Alopecia
  - 1ry.
  - 2ry.
- Non Cicatricial (Non scarring)
  - Congenital
    - Atrichea / Hypotrichea
    - Diffuse
      - TE
      - AE
  - Acquired
    - Diffuse
      - TE
      - AE
    - Circumscribed
      - AA
      - AGA
      - Artefactual Alopecia.
      - Σ Alopecia.

ترتيب الطلاب

A. Cicatricial < 1ry / 2ry.

B Non Cicatricial

. Congenital < Atrichea / Hypotrichea

. Acquired < Diffuse < TE / AE

Circumscribed < AA / AGA / Σ A / Artefactual A

1

Cicatricial (scarring) Alopecia

- All forms of Alopecia in which hair follicles are **Permenantly Lost** & the follicular Epith. is replaced by C.T.

- **When does it occure?** when there is permenant injury of Follicular stem cells.

Classification

Primary

Follicle is the 1ry target of inflamm. attack

(See below)

Secondary

(The Follicle is destroyed indirectly "Innocent bystander")

Secondary Cic. Alopecia

1. Cong.

- Aplasia Cutis Congenita
- Epid. Nevi
- Recessive X linked Ichthyosis

2. Traumatic

- Mechanical e.g surgical scar
- Chemical e.g Caustic Agents
- Thermal e.g Burns.
- Radiation.

3. Infectious

- Viral → H.Z & Varicella
- Fungal → Kerion & Favus
- Bact → T.B
- Protozoal → Leishmania.

4. Neoplastic

- BCC
- SCC
- Lymphoma

5. other conditions

- Sarcoidosis
- Morphea
- Necrobiosis Lipodica. (NBL)

(en coup de sabre)

- Primary C.C. Alopecia. (Acc. to type of Infiltration.)

Lymphocytic

- DLE
- LP — 3 Varieties
  - Lichen planopilaris
  - Graham Little Synd.
  - Frontal Fibrosing Alopecia
- CCCA (Central Centrifugal Cicatricial Alopecia)
- Pseudopelade of Brocq.
- Alopecia Mucinosa
- Keratosis follicularis Spinulosa decalvans. (KFSD)

Neutrophilic

- Folliculitis decalvans.
- Dissecting Cellulitis of the scalp.

Mixed (3)

- Acne Keloidalis Nuchae.
- Acne Necrotica.
- Erosive pustular dermatosis
- Alopecia Parvimaculata.

Non Specific (End stage)

(Idiopathic scarring Alopecia & inconclusive clinical & pathological picture. & include endstage & other Types.)

- Tufted hair Folliculitis.
- Brocq's Alopecia
- Traction Alopecia
- Pressure "
- Trauma "

Discussion of each Type

- DLE
- L.p

→ See the specified section.

Graham little syndrome: - Follicular LP - cicatricial of scalp - Non cicatricial of pubis, axilla

CCCA = Central Centrifugal C.C. Alopecia

(Springl's dis. = Follicular degeneration syndrome)

- Commonest cause of C.C. Alopecia in Blacks
- M : F = 1 : 3
- Pathogenesis: unknown but ± Related to 2 Factors:
  - Anatomical Abnormality of the follicle (premature desquamation of IRS)
  - use of:
    - Chemical Hair relaxers
    - Caustic Cosmetics
    - hot Comb's → كانت بيستخدمها إنسات الزنجير للتسريح وعمل الشعر ناعم.
    - Pomade (مرهم عطرية)

  [تركيبات فرد الشعر زى "الجلاد"]

- the Chic site is: Crown or Vertex; the dis start & remains severe at it & then shows Centrifugal spread
- there may be ass:
  - tenderness & pruritus
  - Baby doll hair (Tufting (منتفش) or hair brush like)
  - pustules & Crusting (d.t super added staph. or immune response to degenerating Follicle). ← وهذه المرحلة ممكن نطلق عليها (Folliculitis decalvans)

Rx if there is:
- No (Marked) inflamm.: Doxycycline or minocycline + superpotent Cs (تشعيرها)
- (Marked) Inflamm. (pustules): 10 ws (١٠ أسابيع): Rifampicin or Clindamycin (300 x2 /day)
  - Maintain: Doxy or Minocycline + Top Cs

(4) كله فرنسي (Pelade = AA)

بروك

Pseudopelade of Brocq

(Brocq's Alopecia)

. Resemble AA

Def: Greatly confusing Term; that may explain: either:
- . specific dis. (1ry form of cic. Alopecia)
- or . Represent the end stage of various other forms of cic. Alopecia & its a diagnosis of Exclusion. * no evidence of inflam.

[برای إستبعاد الأمراض الأخرى]

. AET: . either
- 1ry disorder (Idiopathic) (controversy)
- 2ry disorder (end stage cic. Alopecia e.g end stage DLE or L.P).

. So:
. early < DLE or L.P → late burnt-out scarring Alopecia (end stage).
- early ↓ can be diagnosed as DLE or L.P
- late ↓ No specific diagnosis ↓ "Pseudopelade of Brocq"

. C/P
- . White or slightly pink, cicatricial patches, usually oval or rounded & have insidious onset on vertex of females (M:F 1:3) —Coalescence→ porcelain white slightly depressed (Foot prints in snow) patches on scalp. (middle aged female) (pink - porcelain)
- ⊙ No clinical inflammatory stage., No pustules or Erosions

[Hx:
Active stage: DLE or L.P
C/S
Late stage: No Hx only surgical]

("Histopath.)
. if it represent the end stage of DLE & L.P → Pathology is that of burnt-out Alopecia (All chics of the follicle are lost by scarring) (اخترنا)
- atrophic epidermis

Idiopathic
. in some (1ry) cases: Persistent elastic fibs. around the mid shaft (DD L.P. or DLE → lost elastic fibs.) by acid-alcohol orcein stain

Treatment (2011)

① Burnt-out (No signs of inflamm): Not Necessary

Possible Nor Necessary → Try surgical excision.

② Signs of inflamm. (Activity): → Controversy

- Prednisone + Antimalarial
- Isotretinoin
- Mycophenolate Mofetil

Prednisone + Antimalarial

↕

Isotretinoin.

(5)

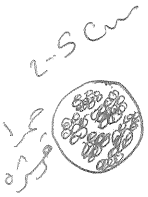
Alopecia Mucinosa (Follicular Mucinosis)

Def. Type of Alopecia caused by deposition of Mucin (CNL component of the ground substance that made mainly of hyaluronic acid) inside H.F.

AET: unknown but may be Mediated (Ht) immune complex & CMI → Mucinous material deposition in hair Follicle & S.G → inflammatory Reaction → Hair loss.

C/P

* 2–5 cm patches, or plaques:
  - Erythematous
  - Scaly
  - studded & grouped follicular papules (Raised Spots) from w Mucinous material can be Expressed. (gelly like material)
  - Alopecia: at First Reversible ⇕ Later: non Reversible.
  - Commonest area: Face, neck & scalp (but any area can be affected).



Types

* there are 4 clinical Varieties (see the table).

Complications Alopecia Mucinosa May progress to MF (MF)

So follow up & Biopsy should be done.

NB: Histopath. & gene Rearrangment Can't differentiate bet 1ry & 2ry Cases. (only clinical).

Treatment (usually not effective):

1. Wait & See approach: for 1ry Cases (Resolve spont.)
2. Cs: Topical, ILs & Systemic
3. Minocycline
4. PUVA & UVA1.

Types of Follicular Mucinosis:

(D)

Paraneoplastic Type

inflammatory Type = [A] Idy (No underlying ass. dis): 3 Types

- Acute (Pinkus) Type.
- chronic Type.
- Urticaria like, Type.

[B] 2ry: ass. e underlying dis. specially MF. (3 Associations)

✳ **Primary acute type** of young persons (< 40Ys, usually children) **(Pinkus type),**	• Usually **localized** (one or a few lesions) on the head, neck and upper arm • Most **resolve spontaneously** within 2 months to 2 years
✳ **Primary chronic type** of older persons (>40 Ys)	• Usually **generalized** (wide spread). • May **persist** or recur indefinitely. • No associated disorders are identified.
✳ **Secondary** (40-70 Ys)	• Usually **generalized** (wide spread), ***may be associated with :*** **1.Benign conditions:** as lupus erythematosus, lichen simplex chronicus, and angiolymphoid hyperplasia. **2.Malignant conditions:** as MF (commonest), Kaposi sarcoma, and Hodgkin disease. **NB:** *In most patients who exhibit both alopecia mucinosa and mycosis fungoides, these conditions appear to develop concomitantly; however, the concern exists that individuals exhibiting only alopecia mucinosa may also be at risk for subsequent development of lymphoma. **3.Drug-induced**: associated with the use of adalimumab and imatinib. (biological)
Urticarial like alopecia mucinosa (rare)	→ . very rare & usually affect ~~Middle aged~~ U . Clp: itchy urticarial papules & plaques on ~~head & neck~~ e in Erythematous ~~(Seborrheic) background~~ . No Follicular Plugging or Alopecia. Course: ~~Waxes~~ & Wane (~~1ms - 15 Ys~~)

(7)

- **Histopathology:**

  - Mucin accumulation in follicular epith & Seb. glands → KCs disconnection
  - Advanced cases: → follicles converted to cystic spaces containing mucin, inflamm. cells & altered KCs.
  - Perifollicular: infilt. of:
    - Lymphocytes.
    - Eosinophils.
    - Histiocytes.
  - ± folliculotropism.

- **How to diff. bet 1ry & 2ry Type:**

  - صعب جداً مفيش فوارق محددة للتفرقة
  - بعض العلماء بيشككوا في وجود ال 1ry Type وبيعتبروا انه عبارة عن :

    "indolent localized form of MF"

  - **Some helpful features that favors the 1ry Type are:**

    1. young age
    2. Solitary plaque
    3. limited No to head & neck
    4. Spont. resolution
    5. No Epidermotropism or atypical Mg lymphocytes.

أهم نقطتين < Spont. resol / No → Epider / → atypical mg ly

Lymphocytic

KFSD

• Keratosis Follicularis Spinulosa decalvans

↓

Cut. Findings + Ocular findings

- at infancy: Localized Keratosis pilaris of face → progressive affection of scalp, Face, eyebrow & Lashes
- at Childhood: Scarring Alopecia in those sites.

- Photophobia
- Blepharitis
- Conjunctivitis
- Corneal inflamation & dystrophy.

Neutrophilic

Folliculitis decalvans.

- Successive crops of pustules, crusts & ± erosions that → Cc. Alopecia. So there are rounded or oval patches of scarring hair loss that shows perifollicular pustules (at edge or center) & Tufted hair (doll's hair) of the plaque.

- AET: unknown; but ± d.t:

  ① Chr. staph. infection (Proved by aspirating staph from the lesions).

  ② Abnormal suppurative Immune Response (± d.t staph) → Follicular destruction.

• Tt

Chronic Antibiotic use (لفترة طويلة)

- Doxy, Clinda or Rifamp.
- Topical CS ± Antibiotics
- supportive: Vit C, Zinc, Selsun-Blue.

Neutrophilic

Dissecting Cellulitis of the Scalp

(Perifolliculitis Capitis abscedens et suffodiens)

. May occur. as an isolated condition or as a part of Follicular occlusion Triad. (رشفوى دكريرى) مهم

- *. dissecting cellulitis of Scalp.
- *. Acne conglobata
- *. Hidradenitis Suppurativa

.AET : . unknown but ± d.t : Follicular HyperKeratosis → Follicular occlusion → Rupture → Neutrophilic & Granulomatous Reaction.

↓

perifollicular pustules & Tender (Nodules) (deep & Boggy) → Abscesses, sinuses → Cicatricial Alopecia

↓ ??

(SCC) ←

. despite that: there is mild pain & Cic. Alopecia & Foul disch. are the Main Complaint.

Treatment

1. isotretinoin ((for 4 ms) ; of choice)
2. Antibiotics
3. Cs (Topical & ILs)
4. CO_2 laser
5. X. Ray, epilation & Surgery

. NB : Follicular occlusion **Tetrad** ??

Follicular occlusion △ + (pilonidal sinus).

Pilonidal sinus

Acne - Keloidalis (Folliculitis Keloidalis)

Def: Chronic conditions characterized by Keloid like papules & plaques on the occipital scalp & post. neck. Common in Blacks.

- Aet: unknown but may be d.t:
  - "Common in Negro":
    1. Curved Hair Follicles → ingrowing of hair (Pierce the skin → foreign body reaction) (Pseudo Folliculitis barbae)
    2. Short Hair cuts (post. hair line shaving by a razor) حلاقة الرقبة (بالموس)
  3. Chr. irritation from short Collars.
  4. Auto immune Process (1ry form of scarring Alopecia)
  5. Antiepileptic drugs.
  6. ↑ No of Mast Cell in occipital Area

- Pathophysiology: Follicular inflamm. → obst. & weakness of the wall → Rupture into dermis → Granulomatous F.B Reaction + scarring → Keloid like scars.

- CIP: . start as: Firm, smooth, erythematous papules → Pustules → healing → Multiple Recurrences → Coalescence → Multiple Papules → healing & Multiple Keloid like scars & CIC. Alopecia. (Abscesses & Sinuses may occur)
  - Keloid along hair line may be >14cm.
- Commonest site: Post. hair line (occiput & upper neck)

Tx

- Prevention (تعليمات)
  - ممنوع ملامسة الرقبة
  - تجنب لبس الياقات الضيقة
  - تنتك الرقبة
  - [الحلاقة والشاوفة ← تذكر ✓]
- Medical Tx
- Surgical Tx (see below)

Ⓐ Papular lesions: →
- Retin A Cream + Topical Cs. Cream.
  (TOPICAL Retinoids أو ... طبعاً يجب بالليل)
- IL Cs (10-40 mg/ml): better after electrodesiccation (or) Cryo (20 seconds Freezing, 1min Freez again) [دوسة بتريد]

Ⓑ Pustular & Crusting lesions:

Topical & Systemic Antibiotics.

Then Topical Cs.

بعد أي نوع علاج (حتى الجراحي) لازم الكريمات تمشي على

RetinA + Cs (Topical)

Ⓒ Large Abscesses or draining sinuses:
- systemic Antibiotics + systemic Cs (loads)

Surgical Tratment

- Small Papular lesion (Failed Response to Medical)
  - Punch excision (To deepest level of the follicle) → inject Wound edges ē Cs (Full conc.) → close ē silk (not nylon) 0-4 sutures. → 1w → ILCs (then) 1/4als.
  - or before excision: Lidocaine 2% (Full conc.) → excise → 1suture → 1w → ILs then 4-6 ws
- Larger lesions
  - Horizontal Ellipse Excision → suture → IL Cs.
- Laser Tx
  - Co2 or Nd:YAG (for excision)

Acne Necrotica (Acne Frontalis).

(Acne Variolliformis)

. Chr. Follicular, necrotizing process may be d.t:

1. Staph.
2. P. Acnes
3. Emotional stress (may participate: causing the pt. to manipulate the follicle)
4. Demodex

. there is: recurrent, small, red follicular papules & pustules close to Frontal scalp Margins → undergo central necrosis → Healing occurs with small pitted scars (variolliform).

Other sites
. Face
. Eye brow
. Trunk.

"Variolliformis"

NB Acne Necrotica & Acne Necrotica Miliaris.

. Similar conditions that cause scalp Folliculitis but A.N. Miliaris differ in:

① More superficial (itchy, crusted, Erythem. Papules → no scarring).

② May affect the whole scalp (not only the frontal line).

. AET ± P. Acnes
staph (severe cases)
Pityrosporum (Malassezia).
Demodex.

Tt ① Treat as Acne:
. Topical: Fucidin, Zinc myCin & Dalacin T.
. Systemic Antibiotics: Tetracycline, clinda.

② "Mild topical Cs ③ Nizoral shampoo.
④ Doxepin (in A. Necrotica). ⑤ Stop oily stylers

Necrotica
miliaris =
of Folliculitis
propionibact-
um Folliculitis

Q. Quiz

Adult
cl, greasy,
lythem papules
is alp

Erosive pustular Dermatosis

(17)

- Condition ch by:
  - Tiny pustules, Crustation, Erosions on ← scalp / Forehead / Temples
  - Elderly women, > 70 yrs.
  - usually develops on top of sun damaged skin
  - usually on Areas of scarring as ← after injury / surgery / skin cancer / H.Z.
  - → cc. Alopecia.
- AET: unknown; but may be related to sun damage & is (Triggered by) minor Trauma (e.g surgery).
- infection: not 1ry cause but ± superadded. (So not responding to Antibiotics)

(HH)

① Crust → Remove ē K. permanganate or Burrow. Sol.

② Inf. → systemic Anti Staph.

③ Main HH:
- Super potent Cs
- Daivonex
- Tacrolimus
- Minocycline (6ws; d.t Anti-inflamm. Actions).
- Cryo.

④ Adjuvant (Maintenance) HH:
- Vit C
- Zinc
- Topical Cs.

Alopecia parvimaculata

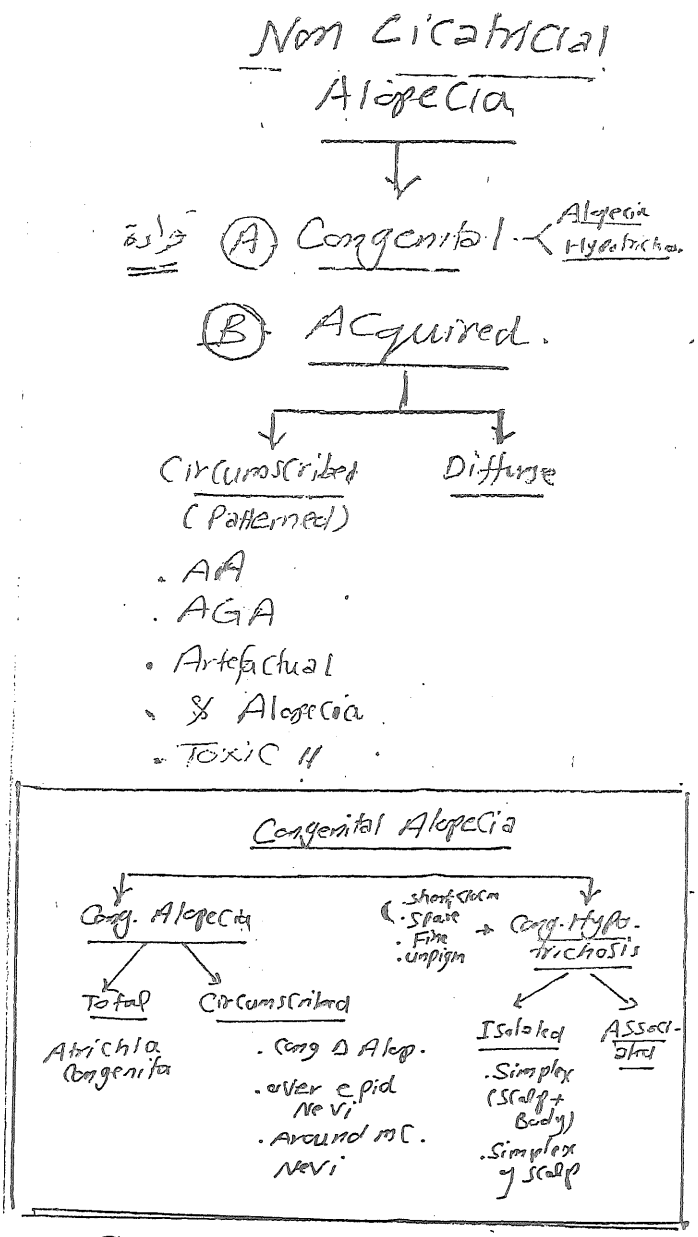
- Epidemic of patchy hair loss affects children living in close proximity.
- oval, rounded or Angular areas of Atrophy & mild inflamm.
- reversible in most cases; However scarring may occur (10–15%) of cases.

- Diagnosis ← Multiple.
  Angular.
  no Hx of inf.
  Not intact skin (c/ A.A)

(NB) . Tufted Hair Folliculitis
(Polytrichia)

- Not a disease But This pattern seen in the end stage of many CIC. Alopecias
- Infundibular epith. of the damaged follicles when heal ⇒ scarring → contraction → aggregation of multiple follicles → doll's or hair brush pattern.
- may be seen in scarring Alop. d.t:
  - CCCA
  - Acne Keloidalis
  - dissecting cellulitis
  - Keloid
  - Pemphigus.

(NB) . Also ± occur NCLY at scalp & legs



Congenital alopecia

Congenital total alopecia (Atrichia congenita)

In this autosomal recessive disease patients are born with hair that falls out between the first and sixth month and is not replaced with no further growth. The eyebrows, eyelashes and body hair may also be absent but more often they show few sparse hairs. Teeth, nails, sweating, growth and intelligence are all normal.

Congenital circumscribed alopecia

This should be differentiated from alopecia areata and acquired cicatricial alopecia. The most common forms are *naevoid*. Epidermal naevi are usually devoid of hair and present as warty or smooth, slightly indurated plaques. A zone of non-cicatricial alopecia sometimes develops around melanocytic naevi. ***Circumscribed non-cicatricial alopecia*** is uncommon. It is the result of hypoplasia or aplasia of a group of follicles. The scalp is clinically normal and histologically shows no change other than a reduced number of follicles. The first hair coat is normal and the patches of alopecia develop between the third and sixth months.

Hypotrichosis Isolated, Ass.

Introduction: The hair follicles are sparse and reduced in size, and the hair shafts are brittle and deficient in pigment. Congenital hypotrichosis may occur as an isolated abnormality or as a feature of hereditary syndromes and associated with other ectodermal defects. Isolated hypotrichosis is autosomal dominant and includes 2 types: hypotrichosis simplex which affects the hair of the scalp and body, and hypotrichosis simplex of the scalp which affects the scalp only. In hypotrichosis of hereditary syndromes, the hair is not sparse but fine and brittle, and is often hypopigmented.

Isolated hypotrichosis: The scalp hair at birth is normal in quantity and quality, but is shed during the first 6 months and never adequately replaced. It is sparse, fine, dry and brittle, and seldom exceeds 10 cm in length. The eyebrows, eyelashes and vellus may be absent, sparse or normal.

Hypotrichosis in hereditary syndromes

- *Hypohidrotic ectodermal dysplasia*: Affected males show hypotrichosis, abnormal teeth and absent sweat glands. Both X-linked and autosomal dominant forms exist.
- *Hypotrichosis with keratosis pilaris*: Besides hypotrichosis there is keratosis pilaris on the occipital region and neck, and sometimes on the trunk and limbs. Nails and teeth are normal.
- *Hypotrichosis, Marie-Unna type*: Two patterns exist. In the more severe form, the child's hair is always sparse and is progressively lost, so that alopecia is advanced by puberty. In the other, milder form, the hair is initially thick and the hair loss only commences in the second or third decade. The eyelashes, eyebrows and body hair are typically absent from birth.
- *Hypotrichosis in disorders of amino acid metabolism*: Fine sparse hair has been reported in phenylketonuria, arginosuccinic aciduria and hyperlysinaemia.

بسم الله الرحمن الرحيم

موضوعنا: # Alopecia Areata (AA)

def. Type of Non-Cicatricial Alopecia Ch BY sudden circumscribed or diffuse hair loss w/ usually reversible & has obscure Aetiology.

Incid: 2% of dermatologic Cases.

Aetiology unknown --> many theories:

1- Genetic factors
2- Immunologic " factors
3- Endocrinological "
4- Psychological "
5- Other factors.

مناقشة كل عامل بالتفصيل

قراءة فقط

(1.) Genetic factors: evidenced by:

- +ve FH in 10-20% of Cases.
- Higher Incid. in identical Twins.
- Significant ass: ē HLA DR4, DR5, DR11
- Ass ē Genetic dis e.g Down.

(2) Immunological Factors: Evidenced by:

a. Ass. Autoimmune dis. e.g L-E, vitiligo (4%) & thyroiditis.

b. Ass. Auto antibodies:
* ANA : 24%
* Anti Thyroid Abs: 25%

c. Ass. AD: (+ve AD in 18% of Childhood AA & 9% of Adulthood AA).

[d] Peribulbar Inflamm. infilt. composed of CD4, CD8 (mainly Thelper 1 cells)

CD4 : CD8 Ratio = 4:1 (NL 1:1 – 4:1)

So Cytokines are ?? Th1 → against MC based Ags

[e] Blood: ↓ Treg Cells & NL T. helper.

③ Endocrinological Factors: evidenced by:

- Ass. Testicular Abnormalities.
- Impaired fertility
- Regrowth of Hair may occur during preg.

④ Psychological Factors:

stress → ppt. of AA by disturbing HPA → endocrinal or other immunological disorders.

⑤ Other factors: (No Evidence):

- CMV
- Septic focus (Dental, ENT) → Reflex irritation of the follicles → hair loss.
- Errors of refract.

- Etiopathogenesis: (Immunoprevilage theory)

- Hair bulb is Immunoprevilage site; in AA it shows HLA I & II expression → attacked by Immune System → CD4 & CD8 infilt. → early onset of Telogen (arrest at Anagen 4).

are retained in clinically hairless scalp of alopecia areata except in very long standing ..... Hair follic...

cases that persist for many years in which there may be a decline in follicle density, possibly associated with fibrosis of the perifollicular connective tissue.

Histopathology

Fix → Horizontal / Vertical sections from advancing border.

- Chic finding: is the presence of Miniature hair structures of early Anagen or Telogen.
- peribulbar infiltrate (2) fibrous Tract remenant beneath the bulb (3) Contains: Lymphoid cells, eosinophils & Melanin. & infiltrate Composed of CD4 (CD4/CD8 = 4:1) (Swarm of bees)

CD4

CD4 : CD8 = 4:1

Clinical picture

1. CIP
2. Clinical types (Varieties)
3. " Classification
4. prognosis & DD.
5. Ass. other changes.

- **CIP**
  - Sex: Equally affect both sexes
  - Age: any age but the peak incid is bet. 20-40 ys. (Cong. cases may be present)
  - Sudden & Complete hair loss in Circumscribed Area in (3) the skin is Completely NL.
    - usually c is covered accidentally.
  - Any hairy area can be affected but the Commonest is: scalp (Other areas: Beard, Moustach, eyebrow, eyelash).
  - in the lesion: Resting hairs may be found; while at the periphery of the lesion there may be an "Exclamation Mark Hair" (thin Proximally & thick distally) that can be pulled out.

6 in Pull

classification

N.B. الشعر ضعيف فوق و قوي تحت (علامة التعجب) غريب جداً

. Exclamation Mark Hair is the Pathognomonic of AA but not always present.

What are signs of Activity of AA ??

& yellow dots by Dermoscope

1. Exclamation Mark Hair.
2. +ve pull test at periphery of lesion. ≥ 6 Exclamat. Mark Hair.

* **Prognosis:**

. always uncertain

. incid. of spontaneous Recovery:

30% → 6 ms.

50% → 1 year.

70% → 5 ys.

. 30% → No Recovery

. 85% → Relapse.

What is AA c̄ Bad prognosis

. NB . Why Alopecia A. Involve the pigmented hair only & why Regrowth of Hair starts unpigmented ??

. this because: there is hypothesis that Melanocytes are the targets for Activated T cells so there is:

. sparing of ~~black~~ white hair → Patient gets white over the Night

. Regrowth of Hair is unpigmented & Fine.

Classification of AA:

* Clinical Types: "شوية ثوابت"

1. Localized AA (one or more patches)
2. Alopecia totalis (affect all scalp)
3. Alop. universalis. (" all body hair)
4. Ophiasis & lateral → Sisiapho — affect the vertex sparing the marginal areas (areas of ophiasis)
5. AA diffusa (diffuse Rapid AA resembling anagen effluvium)
6. Migratory Poliosis
7. Reticular
8. perinevoid
9. linear

(at the marginal scalp i.e. occipital) → Ophiasis

- Clinical classification: (Ikeda classif)

Type	Frequency (%)	Age at onset (yrs)	Duration	A. totalis (%)
I common	83	20-40	< 3 yrs	6
II atopic	10	< 15	> 10 yrs	75
III prehypertensive	4	15-25	Variable	39
IV autoimmune	5	> 40	Prolonged	10

(Totalis in Atopic, longer durat.)

- Associated changes in AA: — Cut., Nail, ocular

1. Eye changes: Post. subcapsular cataract in "Alopecia Totalis".

2. Nail changes

≈ 20% of cases; More common in severe cases.

- include:
  - pitting (Commonest: How to diff. bet it & pitting of PS.)
  - Beau's line
  - onychorrhexis

- usually: Finger nails.

3. Cutaneous:
- Vitiligo
- AD
- CTDs

4. Others:
- Down
- D.M
- thyroid disorder

NB (اسئلة شفوي)

1. Invs For AA →

SPECIFIC INVESTIGATIONS

▶ Consider complete blood count, thyroid function tests, serum B_{12} and autoantibodies as a screen for associated autoimmune conditions.

No routine investigation is normally necessary and the diagnosis is essentially clinical. However, in patients with symptoms or a family history of autoimmune diseases, such as thyroiditis, pernicious anemia, or Addison's disease, autoantibody screening and further investigation may be indicated.

① - CBC
② - Thyroid function tests
③ - Auto Antibodies

---

2. AA ē Bad prognosis (Extent [illegible])

- onset : early (Childhood) onset.
- Course : rapidly progressing.
- duration : longer > 5ys.
- Site : outside scalp (Beard)
- Type : Multiple patches, Totalis, Universalis ophiasis, reticular & Eye brow affection
- ass. → Atopy. Nail pitting. [illegible]

3. DD of AA:

- T. Capitis.
- Trichotillomania.
- Cong Δ alopecia (at birth, Δ at temporal) ← المريض Δ مثلث فارغ من الشعر في جنب الشعر ويكون Cong
- Toxic Alopecia (Hx of Impetigo ± Atrophy)
- Early SLE.
- $\int$ alopecia.

4. Eye brow AA: Some authors ≠ (لا يحبذ) ILCs (Intralesional Cs) to avoid Central Retinal artery occlusion.

5. Eye lash AA: PGFα (Analog) (latanoprost) ® نقط تقفيل ضغط العين

Prostaglandin F2α & analogue e.g. Latanoprost drops

Latanoprost

مستخدم بنوع مرض كلوكوما Glaucoma
وجد انه بيطول الرموش بس بيغمق لون الـ iris

Treatment of AA

(1) Reassurance

> *Introduction:* Leaving alopecia areata untreated is a logic option for many patients. Spontaneous remission occurs in up to 80% of patients with limited patchy alopecia of short duration (less than 1 year). Such patients may be managed by reassurance alone, with advice that regrowth cannot be expected within 3 months of the development of any individuals patch. The prognosis in long-standing extensive alopecia is less favorable. All treatments have a higher failure rate in this group and some patients prefer not to be treated, other than wearing a wig.

- AA: Spont resol.
- 80%
- limited cases
- < 1 y. (short durat.)
- ↓ in 3 ms.

(2) lines of Ht:

FIRST-LINE THERAPIES

- Intralesional steroids
- Topical immunotherapy

SECOND-LINE THERAPIES

- Topical corticosteroids
- Topical minoxidil
- Phototherapy
- Anthralin/dithranol
- Retinoic acid

THIRD-LINE THERAPIES

- Systemic corticosteroids
- Systemic ciclosporin
- Topical ciclosporin ← (قبر)
- Oral minoxidil
- Inosiplex (inosine pranobex)
- Nitrogen mustard
- Dermatography
- Cryotherapy ← جديدة
- Sulphasalazine
- Pulsed infrared diode laser
- Excimer laser
- Aromatherapy
- Onion juice

- Discussion of Ht:

Cs

Intralesional

- 1st line Ht of localized AA (1-2 patches) but can be used for up to 50% of scalp surface area if Tolerated

Topical Cs

- less effective than IL Cs
- Superpotent Cs ± under occlusion 6 d/W for 3 ms.
- S.E folliculitis.

Systemic

- Continuous
- Pulse

intralesional CS

- Triamcinlone acetonide (Kenacort A vial)® = 40 mg/ml

تخفيف الامبول بجابولين
بنسبة ١ : ٢٠ (او) ١ : ٤٠

- Dose : 2-10 mg/ml

.4 → Scalp: .5 mg/ml or [يخفف بنسبة ١ : ٨]
.8 . 10 mg/ml . [١ : ٤]
.16 → Beard / Eye brow : 2 mg/ml [يخفف بنسبة ١ : ٢٠]

- Inject. 0.05 - 0.1 ml ; 1 cm apart every 4-6 ws [intradermal or upper S.C]

(حوالي امبول كامل) . dont exceed 3 ml / session. (Eg vial > 20 mg/session).
(Ep ...) . if no response after 3-6 ms → Stop

- S.E : minimal & Transient eg atrophy.

- NB : (Hydrocortisone acetate) ممكن نستعمل سوليكورتيف
[2-10 mg/ml]

Systemic CS

← continuous / pulse / Minipulse.

Continous

- still debated
- 20-40 mg/d. prednisolone
- disadv:
  1. Refalling if stop.
  2. Not effective in ← AT / AU / ophiasis / Atopics.
- More useful : in ↓↓ ! Progression.

Pulse

جرعة عالية

① . Methyl prednisolone :

- indic. ← early cases < 1y. / area > 30% of scalp / Progressive

(بالامبول)

١ جم يذوب في لتر محلول ملح على مدار ساعتين
يومياً لمدة ٣ ايام [للكبار]
٥٠٠ مجم الجم لكل ١ لتر محلول على مدار ساعتين
لمدة يومين لمدة ٣ ايام [للاطفال]

[نعطي ٤ جرع بينها شهر]

(IJDVL 2013)

② . oral prednisolone : 200 mg [illegible] For 6 ms

③ . oral Betamethasone : (minipulse) 5 mg Twice/w For 6 m. [لو مفيش استجابة ١٠]

سؤال امتحان — مهم أوي

Topical Immunotherapy

(3 Agents) — مبدأ تغيير الخلايا

1. **DNCB**: Dinitro chloro benzene. [Teratogenic so not used]
2. **DPCP**: Diphenyl cyclo propenone. (الأفضل)
3. **SADBE**: Squaric acid dibutyl ester [limited stability] لذا تخزن بثلاجة

- **Mechanism: unknown but ± d.t:**

1 Antigenic competition theory: application → sensitization & irritation → new antigen formation → infiltration by suppressor macrophages & T-suppressor cells → modification of the preexisting infiltration (CD4 : CD8 become 1:1) → Regrowth

2 Other Mechanisms:

- ↓ HLA expression. (See Immunoprevilege theory)
- ↓ Postinflammatory cytokine release by KC.

- طريقة الاستعمال (SADBE) (DPCP) (For children or adults > 10 ys + > 50% involvement)

١- يدهن تركيز ٢٪ على مساحة ٢×٢ سم٢ ويترك لمدة يومين (ثم يغسل) على جهة واحدة من فروة الرأس

٢- بعد أسبوع غالبا يحدث Irritation

٣- يدهن تركيز ٠.٠٠١٪ على كل المناطق المصابة من الجهة اليسرى (مثلا) من فروة الرأس مرة كل أسبوع ويترك لمدة ٤٨ ساعة ثم يغسل ويزاد التركيز تدريجيا (From 0.001% - 2%) الى أن نصل لـ التركيز الذي يحدث (Persistent irritation for 1 day) نثبت عند هذا التركيز مرة كل أسبوع الى أن يحدث تحسن على الجهة اليسرى ثم يدهن على الرأس كلها.

- **S.E**
  - Rash, pruritus, L.N
  - EM, vitiligo & folliculitis
  - Teratogenic
- **C.I**:
  1. Teratogenic
  2. Mg & Blood dyscrasias.

Onset = 3-6 m
Success 30-50%

Topical Irritants

- Anthralin (Dithranol)
- phenol
- Benzyl Benzoate
- Tr. Iodine, Capsicum & Cantharidin (equal parts)

تركيبة رشوة ⇒

Anthralin (in PS. & AA)

- **Mechanism**: ?? but I: ← Cytotoxic / Antiprolif. / Antiinflam.
  - Cytotoxic & antiproliferative → Ht of Psoriasis.
  - Generation of free radicals → ROS have Immuno suppressive & antiproliferative action
- **Methods** either
  - over Night application of lower Conc.
  - short Contact therapy (> 0.5%).

(بولونيا) | AA طريقة الاستعمال في |

نبدء بتركيز 0.5%

- أول ٥ أيام ← دهان ساب لمدة ١٠ دقائق ثم تغسل
- ثاني ٥ أيام ← " " " ٢٠ دقيقة
- ثالث ٥ أيام ← " " " ٣٠ دقيقة

هكذا نزود ١٠ دقائق كل ٥ أيام الى أن يحدث Mild irritation
ونسجل هذا الوقت ونستعمله مرتين يساب طول هذه الفترة.

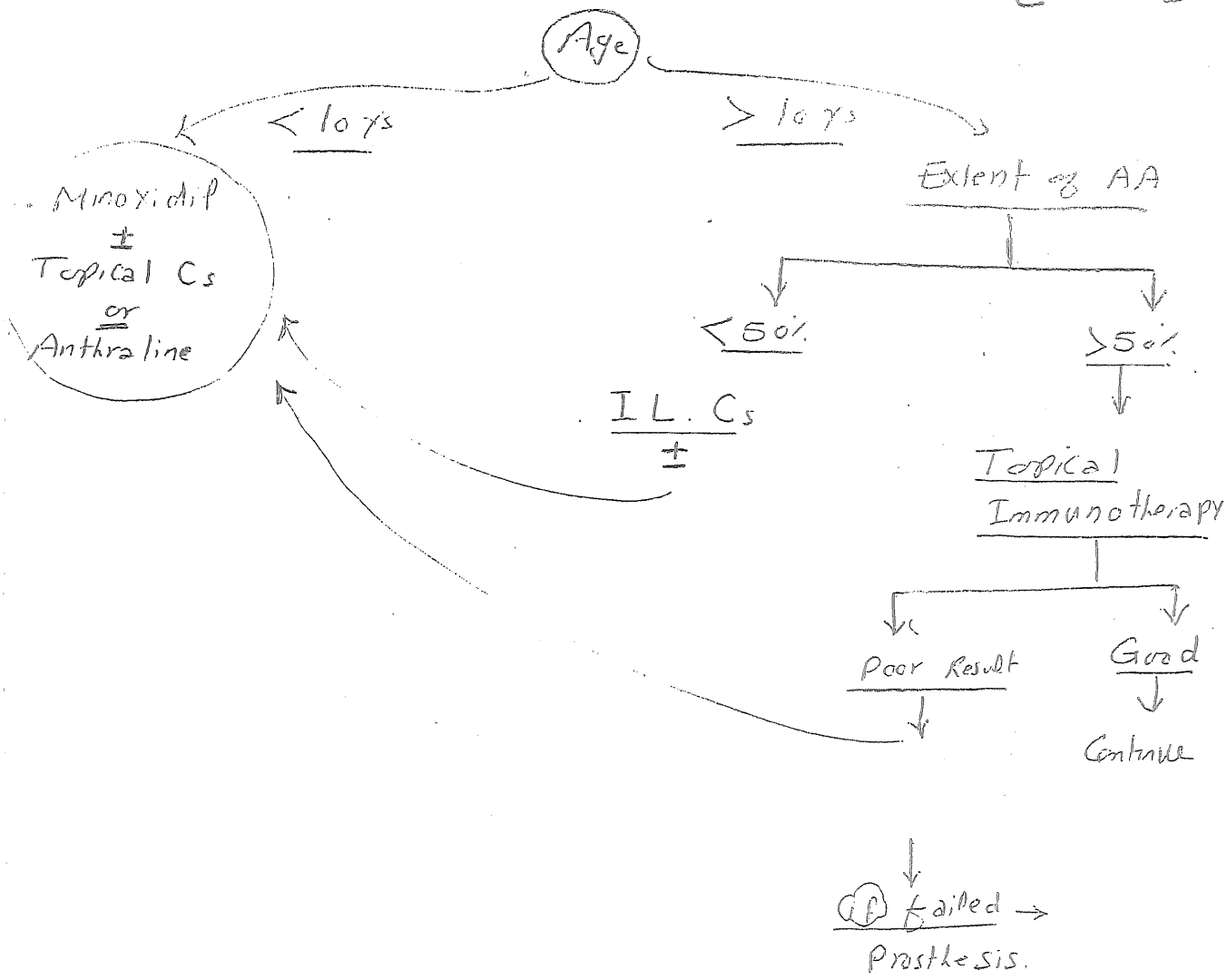
- لو لم يحدث irritation عند ساعة يستعمل تركيز ١٪ بنفس الطريقة السابقة وإذا لم يحدث irritation عند ساعة يدهن مساء ويغسل صباحا (over Night application).

onset of resp. 3 ms & best at 6 ms.

S.E ← CD / Staining of clothes. / Pregnancy (C)

Ht of choice in children < 10 Y.

(E) Treatment Protocol For AA. (American Academy) [2006]



(F). NB Ciclosporine (Topical or systemic):

- Immunosuppressive
- Causes Hypertrichosis.

Androgenic = Androgenetic Alopecia

(← Male Pattern Alopecia)

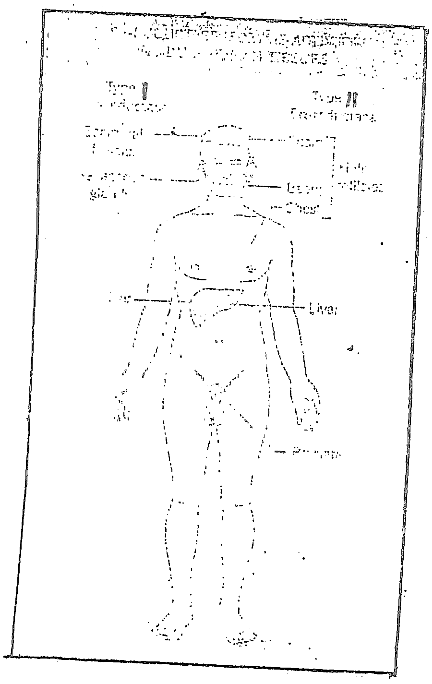
Def: Common type of Non Cicatricial Alopecia That affects both sexes & results from the effect of androgen on genetically predisposed Hair follicles → Hair loss.

So: it Has 2 Factors:

① Genetic factor (but ± can't +ve FH)

②. Androgen induced (مش كافي لوحده)

Effect of Androgen on Hair → See Hirsutism



• Sites of 5α reductase EnZs.

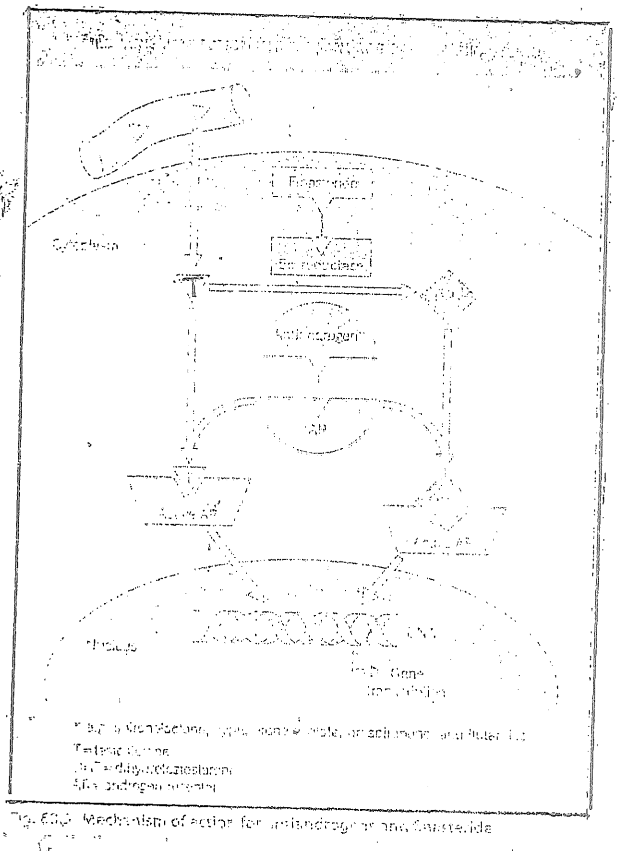


Fig. 63.3 Mechanism of action for antiandrogens and finasteride

N.B.: Seb. gland
- duct → (II)
- lobes → (I)

Pathophysiology of AGA:

① Genetic background: (♂, ♀)
- Not fully understood
- Mostly Polygenic
- Mostly AD.
- ♂s have a stronger "FH".

(B) Role of Androgens (established by):

- No AGA in < Eunuchoids. / Castrated ♂ before puberty
- +ve AGA: if Testost. given to genetically predisposed

عشان كده العلاج بيحيل زرعة تصرفه ماتجي فروة الراس مش تبعه فرق راسه →

- ↑ level of Androgen Receptors (30% ↑) in balding frontal hair follicles than in non balding occipital follicles

• Mechanism of AGA:

- Testosterone is converted to the active form DHT under effect of the enz.

مهم جدا جدا **5α- Reductase**

↓

2 Types (2 Isoenzymes)

Type I	Type II
Formed of: 259 aa	254 aa
PH: Alkaline (6-9)	Acidic (5.5)
Chromosome: 5	Chromosome: 2

Type I

• Sites (اهم حاجة):

1. Scalp Hair follicle < IRS / ORS
2. Seb. Gland lobules. [illegible]
3. Liver.
4. Brain.

NB Type II in dermal Papilla.

Type II

Sites:

1. Follicles of < Scalp / Beard / Chest (IRS, ORS & DP)
2. Seb. Gland duct.
3. Liver,
4. Genitalia < Epididymis / Vas / S.V / Prostate.

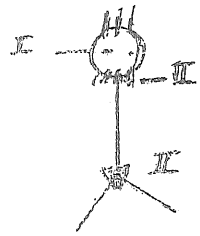
NB: (A) So: Type I: predominate in:

(i). Sab. gland Lobes.

(ii). Scalp Hair

5α-reductase

I & II



while: Type II predominate in:

(i). Seb. gland duct (no role in Acne).

(ii). Hair of: scalp, beard, chest.

(iii). Prostate

Both Types are expressed in:

- Hair follicles
- Seb. glands

(B) The 2 Types are Expressed in ORS & IRS of scalp Hair

DHT

Shortening of Anag. → Less (T) → A: Telo. → Kenogen.

Miniaturization → Thining

DHT (1) → shortening of Anagen → ↑ Telogen

(2) → Gradual ↓ in size of the follicles →

Finally Terminal follicles are replaced by Vellus → Miniaturization → Thinning

Advanced stages → many Vellus follicles disappear.

So the effect of DHT in AGA: is Miniaturization of the Hair follicle. (by effect on DP)

(Terminal → Vellus)

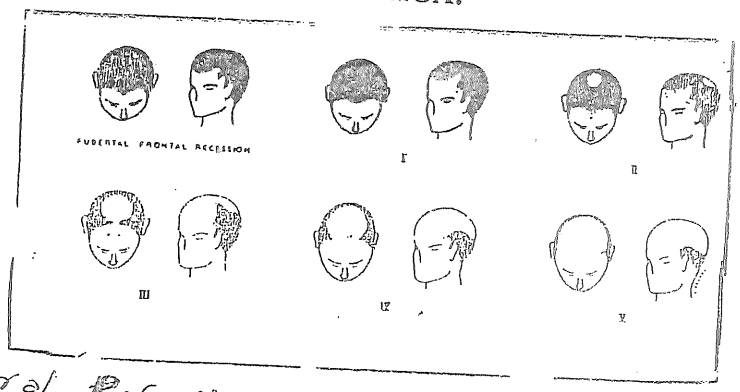
(Pseudo Vellus)

AGA May result from:

- ↑ T. level
- ↑ DHT " (??)
- ↑ Sensitivity of Androgen Rs.

• CIP of AGA

1- Male AGA:



Hamilton / Norwood Grading (only V)

I- frontotemporal Recession

II Same loss on ! Crown

III - V Hair loss on both regions became < Confluent & Extensive.

VI - VII Wide Spread Alopecia Sparing "only" occipital & parietal areas.

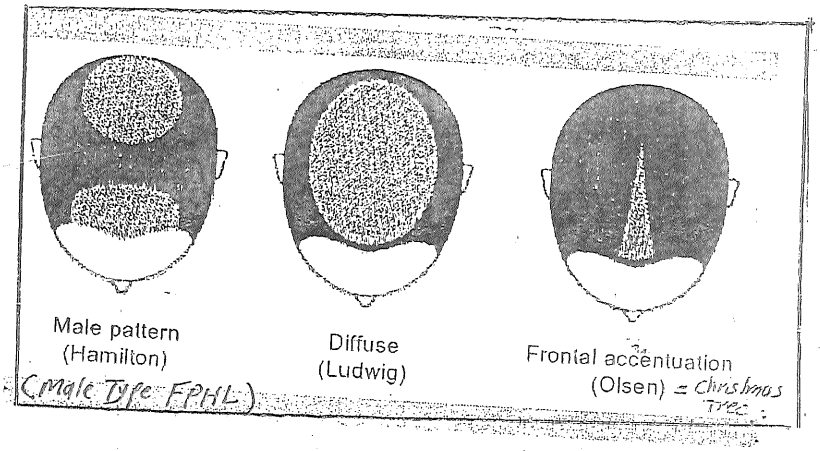
2- Female AGA (Better named female pattern hair loss; FPHL)

Three types of FPHL patterns have been described. [26]

1. Diffuse central thinning (Ludwig type): The diffuse hair loss is concentrated over frontal/vertex (crown) region leading to thinning/rarefaction over central scalp with ***intact frontal hair line*** عبارة مهمة. Ludwig graded it into three stages depending upon whether the central thinning is mild (stage I), moderate (stage II), or severe, that is, near-complete baldness of the crown (stage III).
2. Frontal accentuation (Olsen type): It leads to widening of central parting line and thereafter to chrismas-tree pattern with ***intact frontal hair line***.
3. Frontotemporal recession/vertex loss (male pattern/Hamilton type): It leads to recession of frontotemporal hairline or bitemporal recession and/or thinning at vertex (similar to male AGA so graded as it).

1 cast Common ←

The first two types are common and the third type is seen infrequently. The first type is often confused with CTE.



(Adult)
1. Temporal Recession (Hamilton) occur in virilizing Condition.
2. FPHL: ± clinically apparente Superimposed TE.

All → Central Thinning (Frontal / Vertex or Crown) ← Diffuse (Ludwig). Frontal recession = Widening of central parting line (Olsen) ✓ christmas

Bitemporal Recession

Investigations For AGA: ④

- Trichogram & Dermoscopy
- Lab < if SAM / if TE
- Biopsy

① **Trichogram:**

- ↑ % of Telogen (1:5) (Telogen : Anag.) → loss
- ↑ % of Vellous (<4:1) (Term. : Vellous) → Thinning
- ↑ Kenogen

[NL 8:1]

② **Androgen level:** No Routine Inv. For females ē AGA Except if there are signs of Hyperandrogenism (CAH, Hirsutism, Irreg. menst.) ⟶ See Hirsutism Inv.

③ **Both AGA & TE** (Frequently exist): Exclude Causes < Fe. level, T3, T4, Nutrition

④ **Scalp Biopsy:** to diff. bet AGA & CTE.

- (i). Miniaturization ē ↑ V/T ratio
- (ii). ↑ T/A ratio (1:5)
- (iii). peri bulbar infilt
- (iv). fibrous Tract remenant (Stream) beneath the miniaturized Hair

- **NB**: "pull test" usually Negative [illegible]

⑤ **Dermoscopy**:

- Miniaturized Hair
- Peri bulbar casts.

NB - AGA is ass ē ↑ risk of
- CAD
- IR
- Other Cut. sign of CAD

Treatment of AGA

- **Medical**
  - **FDA approved** (2)
    - Minoxidil
    - Finasteride.
  - **Non FDA approved** (3)
    - (1) Dutasteride
    - (2) Androgen Receptor Blockers e.g Aldactone & CPA (in ♀)
    - (3) Latanoprost (off-label)
- **Surgical**
- **Laser (LLLT)**
  - low-level Laser light therapy.
  - (Med.)
  - (FDA) approved

NB
- Cyproterone Acetate (CPA)
- 50-100 mg/d in AGA
- 2 mg/d For Hirsutism.

• Surgical Rx

- **Hair Transplantation** either By
  - Autografts
    1. Punch (4mm)
    2. Micrografts
  - Flaps
    Rotational from parietal to frontal.
- **Scalp reduction** either By
  - Surgical Excision
    Elipse excision From Vertex
  - or Tissue Expanders.

• Low-level laser light therapy (LLLT) (2012)

"Diode"

* Low-level laser light therapy, in particular a "red light" hairbrush–like device has been marketed as an over-the-counter technique for hair growth. In a double blind, sham-device controlled, multicenter, 26-week trial, 110 patients in the active treatment group who completed the study showed a significantly greater improvement in overall hair regrowth than did the sham group.[29] Marketed as the HairMax LaserComb, it has obtained 510K FDA approval for use as a medical device. Note that this approval refers to safety rather than actual efficacy and that the data required for medical devices are quite different from those required to demonstrate the safety and efficacy of drugs.

• Others:

1. Latanoprost o.i.n & Bimatoprost. (PGF2α analogue).
2. Bioengineered KGF VEGF.
3. PRP

Minoxidil

[No Hormonal
Nor Immune Suppressive]

- Mechanism:
  - unknown . but ± d.t:

↓EGF ↑VEGF

1. Potassium Channel opener: Minoxidil → M. sulfate → opening of K. Channels → ↓ intracellular Ca+ → -- EGF → Hair growth. (Ca++ Normally ++ EGF to -- Hair growth).

2. Other Mechanisms:

++ KC → Mitosis, survival

- direct mitogenic effect on KCs
- ↑↑ KCs survival
- VD (usually not a role)
- opening of K. Channels → ↑ ATP → ↑↑ Adenosine → ++ VEGF → ↑ Hair growth.
- ++ PGs in Dermal Papillae: → ↑ Hair growth.

(Latanoprost ل طريقة تشبه)

- Indications:

FDA approved

- AGA (♂ & ♀)

لازم يكمل "مريض" لمدة الحياة وإلا لو قطع عند الحالات أو الجرعة ← Hair loss.
في خلال ٣ شهور

- 2% → Female
- 5% → Male.

| 5% in ♀ is effective but ↑ Hypertrichosis.

Non FDA Approved
(off label uses).

1. AA (... الخلط)
2. Prehair transplantation; يفضل استعماله قبل زراعة ؟؟ improve function of suboptimal follicles & may optimize the Transplanted Follicles survival c̄ subsequent use.
3. Cong. Hypotrichosis
4. loose anagen Synd.

FDA for ♀ FDA for men

Concentrations: 2% & 5% (5% More effective).

• dose: Not > 25 drops twice / day (1 ml twice / day) (= 50 mg / ml)

• S.E: (usually well Tolerated):

<5% → Hypertrichosis (distant)

Retinopathy (2011) 7% → irritation & CD (± d.t < propylene Glycol) → ↓ by Foam prep (Minoxidil ...)

↑ rare: Headache, chest pain & ankle edema.

onset of Results: 3 - 4 months. [Peak: 6-8 ms] ("أطرش") (لازم يمشي عليه على طول والا هيحصل سقوط ...)

its success was noted in: (Early, mild - mod cases)

* Early cases < 10 ys
* Limited extent < 10 cm^2
* Pre Ht hair density > 20 hairs / cm^2
* More effective on Vertex area.
* More effective in Women.

Clinical Tips: (4) (Pregnancy C)

① لازم تفهم المريض ان في بداية الاستعمال هايبدء يسقط الشعر (Early Terminatn of Telogen to initiate Anagen)

② استعمال ↑ (Retin A) قبل الاستعمال يزيد من مفعوله [بيدهن [أكنيتين A] لوسيون قبل الإجابة بنصف ساعة مساء كل يوم]

③ التجنب حدوث: Facial Hypertrichosis: تستعمل قبل النوم بساعتين (علشان متتمسحش في المخدة) يتم وضع فوطة على الوجه قبل الاستعمال المستمر للوجه ...

④ Avoid use on → Abraded or inflammed or Moist scalp → ↑ abs. → ↑ S.E
→ greasy gels or oint → ↓ abs.

Finasteride

♂ [illegible Arabic]
2013 FDA warning → Irreversible ED.

- has a specific competitive inhibitory effect on the Type [?] 5 α reductase enz. (high conc. in < inner root sheath / Prostate) → ↓ serum & tissue DHT.

[Category X]

- C.I: in pregnancy. ?? because ↓ (FDA)

*** Doses:**
- BPH: 5 mg 1d
- AGA < ♂: 1 mg 1d / ♀: ?? → Controv.
- Hirsutism & AV: 2.5 mg – 5 – (less effective) ↓ (~)

Sexual ← S.E:
1. ↓ libido.
2. ED.
3. Ejaculatory disorders.
4. affect sperm motility
5. Teratogenic: feminizat of ♂ fetus. (Category X)
6. ↓ PSA by ≈ 50% (baseline assessment indicated in Male > 50 ys)
7. Depression.
8. ± Gynecomastia
9. Hepatotoxic (CH- EFBKm dis. 2012)

rare < 2%, reversible ē stop or ē continuation of the drug.

FDA 2013 → Irreversible ED

NB - More effective in:
- Men: 18 – 41 ys.
- Mild – Mod cases.
- Crown scalp > Frontal
- Combinat. ē Minoxidil.

Finast. ↓ % of Cancer Prostate

NB - Effective in ♀ < AGA (Controversy: 2.5 mg 1d) / Hirsutism: 2.5 mg – 5 mg 1d)
- C.I for pregnant ♀ to touch the crushed tablets.

Dutasteride

(Finast. [illegible Arabic]; Not FDA: [illegible Arabic] Approved.)

- Inhibitor for both Type I & II 5α reductase
- Effective in Rx of Male AGA (JAAD 2006)

Artefactual Alopecia

1. Traction Alopecia
2. Trichotillomania.
3. pressure Alopecia
4. CCCA.

- **Traction Alopecia** (جاذبة)

- Alopecia d.t prolonged Tension on hair (traction)

- **C/P**: usually affect periphery of the scalp specially, Frontal, Temples & above the ears.

- **Clinical Types:**

1. **Marginal Type**: d.t **Pony-tail** style
   → Traction along Frontal line

2. **Brush-rollers Alopecia**:

3. **Brush Alopecia**

4. **Hair-Weaving**

- **Pressure Alopecia:**

- **NB** Artefactual Alopecia ≠ Cic. Alopecia.

Trichotillomania

(. Tricho = Hair
. Tillo = Pulling
. mania = Madness)

Def: Obsessive Compulsive disorder in which patients are driven to pull their own scalp Hair or less Commonly, their Eyebrows, Eyelashes, & Even pubic hair.

- **Epidemiology** (Girls)
  - **Age:** Children > Adults (5-12 Y.) (7 : 1) [Typically adolescent girls]
  - **Sex:** ♀ > ♂ (♂ : ♀ 1 : 4-7)

- **Etiopathogenesis:** the Etiology in:

- **Children:** Anxiety about:
  - Parent relationships.
  - School difficulties.
  - Body Image
  - Sexual Abuse

- **Adults** « Marked psycho-Pathology »
  - Anxiety.
  - depression
  - Obsessive-Compulsive disorders.
  - Panic attacks.

↓

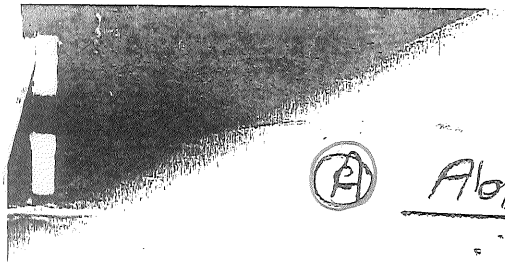
(E). ↑↑ sense of Tension → pulling of Hair → relief of tension.

NB: 2 Types

Non focused Pulling
- Automatic, Non Intentional & habitual Pulling
- No awareness of pulling
- No ↑ Tension

focused Pulling
- Intentional (aware) act to Combat -ve emotional stress (as anxiety & Anger)

Trichotemnomania: Compulsive Cutting or Shaving (not pulling)



C.1P

(A) Alopecia: ← Twisting pulling or picking. → patch or diffuse ē sparing of.

- usually at scalp but any other area can be affected [Eye Lash & brow]
- Patch y/n or diffuse (Full) ē sparing of hair lines (occiput).

the patch ch isy.
- bizzare shaped
- irregular border
- Containing hairs of varying lengths.

(B) Associations of Trichotillomania:

1. Other impulse Control problem e.g ← Biting picking.
   - Nail & lip biting
   - Skin & Nose picking
2. Trichophagia: "سحر، سحر"
3. Trichobezoar: Hair forms Balls in the stomach ē Nusea, diarrhoea, Vomiting & int. obst.

4. Inf. & Folliculitis.
5. Cic. Alopecia. ← يتحول إلى

NB. Compulsive habit of Nail (biting) called onychophagia while picking called onychotillomania.

H ??

Diagnosis

(A.) Clinical <
- Criteria (بودينا 992)
- **Window test:**

بنحلق منطقة (٢×٢) سم الشعر ونلاحظ نمو الشعر جيدا في خلال أسبوعين
هالمنطقة بينمو طبيعي لأنه بيبقى قصير وصعب ان يشده فما يعرف يشده

(B.) Pathology: (Trichomalacia احفظ كويس):

مهمة جدا في الـ pathology

- Most hair is in Catagen or Telogen.
- **Hair follicle** *:
  - Empty or
  - **Trichomalacia**:
    - Shaft: "Fragmented" into dark bodies.
    - Bulb: "Twisted" ē Necrotic Keratins & melanin incontinence.
- **Severe cases:** <
  - RBCs outside the follicles.
  - Cleft (separation) bet hair bulb & the surrounding Collagen

---

- **DD:** (of localized or "patterned Alopecia")
  1. AA
  2. Traction Alopecia.
  3. Pressure " (Iatrogenic)
  4. Toxic Alopecia (following inf)
  5. loose Anagen Hair Synd ??
  6. Moth eaten Alopecia (S).
  7. T. Capitis.

- **N-Acetyl Cysteine (NAC) (Mucolytic):**
  - Mech. modulate Glutamate & Dopamine neuro. Transmission.
  - Dose: 1200 - 2400 mg/d (40-70 mg/kg)
  - Sachets: < 200 / 600

- **Tt** (تحول لطبيب نفسي, No specific effective Tt approach).

Ⓐ **Antidepressants:**
- SSRIs
- Imipramine.

Ⓑ **Others**
- Behavioral therapy
- Hypnosis.

Telogen Effluvium (TE)

. **Def.** Excessive, diffuse shedding of NL. Telogen club hair. Commonly occurs 3-5 m following a stressful condition. ω ± reversible ē in < 6 ms (ATE) or has > 6 m durat° (CTE).

. **Pathology:**

. Introduct° → see Hair Cycle. (تكتب)

. in TE: There are loss of ≃ 150-400 hairs/day (instead of loss of 50-150 hairs/d)

. **Possible Mechanisms:**

1. Premature Conversion of Anagen to Telogen: as in: ATE (تحت إشراف الحمل)

2. (Prolongat° of Anagen) .NB: Pregnancy → Prolongat° of Anagen —الولادة→ Premature Conv. of Anagen to Telogen → Shedding

3. Shortening of Anagen: as in CTE & AGA, AA (طالما عمرها بقى قصير)

4. Shortening of Telogen: ē Minoxidil (شامبو يزود السقوط بداية الاستعمال)

. **C/P of TE** < Shedding (common) / Thinning

1. Diffuse Shedding (الشكوى) → . الشعر بينزل كتير. / . الشعر بيطلع كتل chunks / ع الفرش - المخدة - الحمام - الأكل.

2. Diffuse Thinning (Not Central As AGA) → الشكوى: . فروة الشعر باينة. / . ديل الحصان بقى رفيع عن زمان.

   . ATE → Common
   . CTE → uncommon.

3. Associations:
   . Trichodynia: minimal hair tract → pain is cute ± d ass. anxiety or depression
   . Mod. - severe Bitemporal recession (in CTE)

CAUSES OF TELOGEN EFFLUVIUM
• Shedding of the newborn (physiologic)
• Postpartum (physiologic)
• Chronic telogen effluvium (no attributable cause or illness)
• Postfebrile (extremely high fevers, e.g. malaria)
• Severe infection
• Severe chronic illness (e.g. HIV disease, systemic lupus erythematosus)
• Severe, prolonged psychological stress
• Postsurgical (implies major surgical procedure)
• Hypothyroidism and other endocrinopathies (e.g. hyperparathyroidism)
• Crash or liquid protein diets, starvation
• Drugs
Retinoids (acitretin, isotretinoin)
Discontinuation of birth control pills
Anticoagulants (especially heparin)
Antidepressants
Lithium
Amphetamines
Antithyroid (propylthiouracil, methimazole)
Anticonvulsants (e.g. phenytoin, valproic acid, carbamazepine)
Heavy metals
β-blockers (e.g. propranolol)

Table 56.1 Causes of telogen effluvium. Some authors also propose vitamin B_{12} or iron deficiency as causes.

- Diagnosis of TE ← Clinical, Lab, Biopsy.

A. Clinical Diagnosis

① Pull test: >4-6 club hairs → TE
>2-3 " " (in freshly shampooed) → TE

② Pluck test (Trichogram): Calculate Anagen / Telogen.
- NLLY: Anagen 90-95% & T. 5-10% (1:10)
- TE: Telogen: ≥ 15-20%

③ Clip test: Calculate Anagen / Telogen ratio.

④ Comb test (Serial 1 min Count): in TE; Telogen Hair may be > 100. (in Each time of Combing).

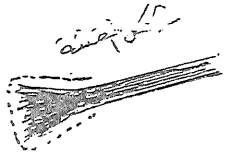
(Vertex to Frontal)

⑤ Timed Shed Hair Count (if pull test is +ve → No need). ④

Telogen → in TE > 150-400 /d.

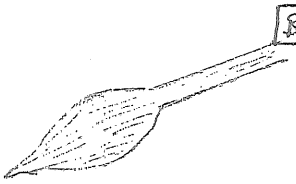
NB • Shape of Hairs: (Mic features):

[A]. Anagen:



- Broom-stick like
- Pigmented
- Surrounded by Gelatinous root.

[B]. Telogen:



- Club. shaped
- depigmented
- No Gelatinous root.

MCQ [C] loose Anagen Synd → Ruffled Cuticle"

[D] Dystrophic Anagen: Thin & Taper proximally

B. Lab investigations

- Lab inv. Should be based on < Hx & Ex
- If the cause is unclear:
  - Thyroid FTs.
  - Chemistry panels
  - ESR
  - Hematocrite value.
  - S. ferritin → (Should be at least 50 ng/dl)

NB
Ferritin
- ↑↑↑ in Chr. or Bleed Inf. (Acute phase reactant)
- ↓↓↓ in: Hypoth. & Vit. C def.

(CRP) ←

CNL < ♂: 30-400 ♀: 15-200

• DD of TE = Diffuse Hair loss

Differential diagnosis of TE generally includes FPHL, CTE, and rare cases of diffuse AA. The differentiating features of TE, FPHL, and CTE are enumerated in the table. Abrupt onset diffuse AA with diffuse thinning and positive pull test may mimic TE, but the presence of exclamation point hairs, dystrophic hairs, circumscribed alopecia at other hair-bearing body areas, nail pitting, yellow dots on dermoscopy, and presence of peribulbar inflammatory lymphocytic infiltrate (swarm of bees) [18] clinches the diagnosis of AA

[DD of diffuse Hair loss] لحبب سؤال الامتحان (5)

Telogen effluvium (TE)
Diffuse type of female pattern hair loss (FPHL)
Chronic telogen effluvium (CTE)
Anagen effluvium
Loose anagen hair syndrome
Diffuse type of alopecia areata
Congenital atrichia, congenital hypotrichosis, and hair shaft abnormalities (hair breakage, unruly hairs)

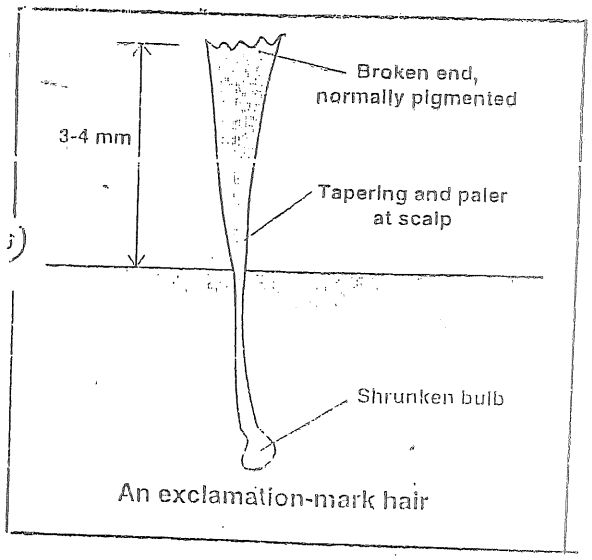
- Treatment of TE:
  - No specific therapy (Hair regrows ✓ in Few ms) (Recession ↓ (6m))
  - # of the cause
  - Eat balanced diet.

• Anagen Effluvium (AE)

- Causes:
  - Cancer Chemotherapy → Cyclophosphamide & Colchicine.
  - Other causes:
    - Trichogram:
      - NL T.
      - ↓ A.
      - ↑ dystrophic Hairs.
    - 1. Pemphigus
    - 2. LE & AA
    - 3. lichen planopilaris (Lyp)
    - (Eg) 4. loose Anagen synd: (AD)
      - blond girls: 2-9 ys.
      - improves ē Age.
      - defect on cuticle (eg IRS): instead of firmly anchoring the shaft it folds back like "rumpled sock" → Ttt: minoxi. Easy extraction.

Clin. blond girl ē Hair:
- Curly
- Uneven length
- Patchy
- Uncombable
- Shedding

- Trichogram:
  - ↓ Anagen
  - TR → 70



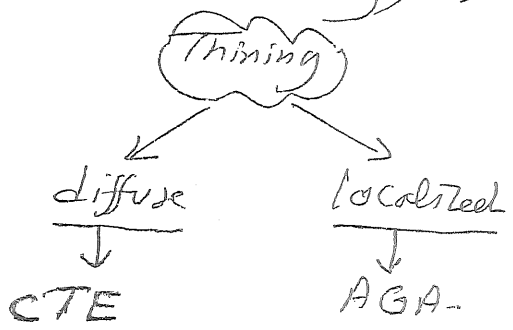
- Treatment of Anticancer therapy Induced Hair Falling :

قبل البدء بالعلاج < pressure cap around scalp
Cold compresses at the ".

Complaint of Hair Falling

1- loss (Hair falls at its roots)

2- Thining (little loss but marked Thining) → visible scalp



Features	Telogen effluvium	Female pattern hair loss	Chronic telogen effluvium
Cause	Underlying trigger, high fever, parturition, etc.	Multifactorial, hereditary, hormones, age	Idiopathic
Onset	Abrupt	Gradual	Abrupt
Shedding	Excessive, diffuse, and generalized	Minimal	Excessive, alarming (hallmark)
Scalp appearance	Diffuse hair loss	Normal or with sparse hairs at central scalp area	Diffuse hair loss
Thinning	Diffuse thinning	Central thinning with or without widened central parting line	Absent, if present, it is all over. h/o reduced ponytail volume
Bitemporal recession	Absent	Mild to moderate and only in male type FPHL, which is uncommon	Moderate to severe and common
Miniaturized hairs	Absent	Present (key feature)	Absent
Hair pull test	Strongly present throughout the scalp	Usually absent, if present, only at central scalp	Present throughout in active phase
Trichogram	Significantly reduced anagen: telogen ratio	A:T ratio is normal or slightly reduced	Reduced A:T ratio in active phase
Dermascopy	No variation in shaft diameter	Marked variation in shaft diameter Miniaturized follicles (hallmark)	No significant variation No miniaturization
Biopsy	Increase in percentage of telogen hairs (11–30%), terminal:vellus (T:V) ratio normal, no miniaturization	T:V reduced (<4:1 is diagnostic)	T:V ratio normal (8:1)
Course	Self limited, event specific	Gradually progressive	Prolonged and fluctuating

- Excessive Hair growth

- Hirsutism
- Hypertrichosis

Hirsutism

♂ (androgen dependant sites) → Male-Pattern growth

♀

def → Women with Excessive growth of Terminal Hair (Thick, long, Pigmented, coarse hair) affecting the Androgen dependant sites.

- NB < Androgenic sites → 1. Terminal Hair, 2. Androgenic sites
  - Androgenic sites → upper lip, chin, lower abd. & inner Thighs

Hypertrichosis: Excessive growth of (Vellus or Terminal) Hair in the Non Androgenic sites above the Normal for the age, Sex & Race.

Etiopathogenesis:

(A.) Sources of ♀ Androgens
(B) Effects of Androgen on H-follicle
(C.) Causes of Hirsutism.

A. Androgen secretion in ♀

↓

Hypothalamus

- Corticotropin releasing Hormone (CRH)
  - ↓
  - ++ pituitary
  - ↓
  - ACTH
  - ↓
  - ++ Adrenal gland →
    - Androgen
    - Cortisol.
- GnRH
  - ↓
  - ++ pituitary
  - ↓
  - FSH & LH
  - ↓
  - ++ ovary →
    - Estrogen (Prog.)
    - Androgen.

- **Adrenal Androgens:**
  - DHEA & DHEA-S
  - ↓ Test. (small amount)

- **Ovarian Androgens:**
  - Before Menopause: Androstenedione.
  - After Menopause: Testosterone.

↓

DHEA, DHEA-s & Androstenedione

↓

Testosterone ← - - - - - - • in serum:
- 50% bound to SHBG
- 48% bound Albumin
- 2% Free.

↓ 5α reductase in H.F (I & II)

DHT

↓

B. Effects of Androgen on H. Follicles of Body

↓

Converts Vellus Hair to terminal Hair → (Hirsutism)

↑ معكوسين عكس بعض ↓

(NB): effect of T. on scalp H. Follicles. — الميكانيزم في الصلع

1. Converts Terminal Hair to Vellus Hair → (AGA)
2. Shortening of Anagen → ↑% of telogen → shedding.
3. Prolongation of Kenogen lag phase → empty follicle.

↓ All

(AGA.)

(NB 2) All Terminal Hair is Androgen dependant (except) ← Eye lash, " Brow, scalp.

(N.B.) T. Metabolitized to 17 Ketosteroids —Ex→ Urine.

So Hirsutism may result from:
- ↑ Androgen level (rare).
- ↑ Sensitivity of Rs
- ↑ 5α Reductase Activity. II

(C) Causes of Hirsutism

1. Familial (Racial).
2. Idiopathic (End organ Hirsutism).
3. Genetic Syndromes:
   - Achard Thier's Synd.
   - Turner's Synd.
4. Drug induced. < Androgen / [illegible]
5. Pituitary disorders:
   - Cushing
   - Acromegaly
   - Hyperprolactinemia.
6. Ovarian disorders:
   - PCOS (Stein-Leventhal Synd)
   - Virilizing ovarian Tm.
   - Hyperthecosis (in post menopausal either → Hyperthec. / Tm)
   - Insulin Resistance states. (CHAIRAN)
7. Adrenal disorders:
   - CAH.
   - Cushing dis.
   - Virilizing adrenal Tms.

> The Most Common Causes:
> - Idiopathic
> - PCOS

مناقشة كل سبب بالتفصيل
(Pathophysiology)

1. Racial:
- Hirsutism common in: ← الشرق الأوسط، روسيا، جنوب أوربا
- Less common in: ← الهند وآسيا
- (FH)

2. Idiopathic (End organ Hirsutism): (Ch BX)
- onset at puberty.
- Course: Stable ← مستمرة
- AET: may be d.t ← ↑ activity of 5α Reductase. / Hyper sensitivity of Receptors to NL level of Testest.
- Rx: Androgen Receptor Blockers ⊕ enz. inhibitors.
- NO = كل حاجة عندها كويسة
  - FH
  - Abnormal Hormonal profile
  - other Androgenic manif. (e.g AGA).

3. Genetic:
- Achard Thiers Synd: نادرة
  - Obesity
  - DM
  - HTN
  - Hirsutism
  - osteoporosis
  - NL urinary Ketosteroids.

(45XO) Turner Synd: one X chromosome is damaged → under feminization & Hirsutism & infertility). عالية وظاهر لما تولد عندها سويه

4. Drug induced:
(A) Drugs ē Androgenic chem:
- Testosterone
- Danazol
- Anabolic steroids.
- Progestins (present in some OCPs)

(B) others
- Aldomet
- Reserpine
- Metaclopramide.

↑ Function of supra renal gland → ↑ Cortisol Secretion

5. Pituitary Causes :

① Cushing dis. (↑↑ ACTH → ↑ Androgen, ↑ Cortisol → ↓ SHBG → ↑ Free T)

(Cushing)

② Acromegaly. (GH → affect ♀ follicles directly) direct ++

③ Hyperprolactinemia:

. Causes : See infertility ??

. Mechanism: unKnown but ± d.t :

ⓐ prolactin

- ++ Adrenal Androgens
- -- Act. 5α reductase (weak)
- -- FSH & LH.

ⓑ associated PCOS.

---

⑥ ovarian Causes :

. PCOS
. Virilizing ovarian Tms
. Hyperthecosis
. Insulin Resistance.

NB LH : FSH > 2 → Suggestive of PCO.

(HH of SKm dis. 2012).

(A) PCOS

(Through the Name; ± occurs in ♀ with ovarian cyst)

How to diagnose PCOS. ≥ 2 of 3 after exclusion of ↑ PRL & ↑ FSH levels

(1). oligomen.
(2). Hyperandrogenism (clinical / lab)
(3). U/S

USA Criteria

1. Amenorrhea or oligomenorrhea [<8-9 cycles/y] or >35 ds cycle.
2. Hyperandrogenism (Clinical (Acne, hirsutism) or Lab)
3. Exclude other causes of menstrual irregularities & Hyperandrogenism. (Hypothyroidism)

- Complications of PCOS ← DM, HTN, CV dis. [Metabolic Synd. Infertility. Endom Cancer]

European Criteria

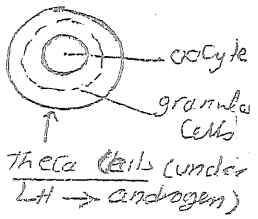
1. as USA
2. as USA
3. U/S diagnosis of PCOS Either:
   - ≥ 12 Follicles in at least one ovary (measuring 2-9 mm in diameter) **or**
   - Total ovarian Vol. > 10 cm^3

(B) Virilizing ovarian Tm: e.g.

- Leydig Cell Tm
- Hilar " "
- Theca " "

(C) Hyperthecosis:

- Diffuse Hyperplasia of Theca cells of Graafian Follicles & presence of these cells in the ovarian stroma → ↑ Androgen production



- Is it a distinct entity or part from PCOS?? Unknown but it differs from PCOS in that it can occur postmenopausal while PCOS in the Reproductive (II. Clin: as PCOS but ± severe virilization & Postmenop. > pre) Period.

(D) Insulin Resistance. (IR)

- IR → ↑ insulin level → ↓ SHBG (sex H. binding globulin) → ↑ Free T.
- ++ GnRH → ++ ovarian Androgen. (FSH, LH) Direct effect on ovary
- ± Ass-e: (1) obesity (2) AN (Acanthosis Nigricans) (3) PCOS — HAIRAN synd.

HA	Hyper Androgenism
IR	Insulin Resistance
AN	Acanthosis Nigricans

7. Adrenal Causes

A. CAH [Cong. Adrenal Hyperplasia]
B. Adrenal Tms (virilizing)
C. Adrenal Cushing

• CAH (Adrenogenital Synd).

Def: AR, Synd, Caused by deficiency of one or more of Enzs. responsible for Cortisol Synth. → ↓ Cortisol production →
① Addison's Manifs
② Lack of -ve feed back Mech. of Cortisol on Ant. pituit. → ↑ ACTH → Adrenal Hyperplasia.
③ shift of pathway from Cortisol side to Adrogen side → Hyperandrogenism.

(See diagram)

Commonest 3 Enzs:
- 21 Hydroxylase
- 11 "
- 3 BHSD

• CIP of CAH

Prenatal
- Female pseudohermaphrodite.
  - small vagina
  - Big clotris
  - Big Fused Labiae Majora (simulating open scrotum).

Prepubertal
- ♂: precocious pseudopuberty (precocious puberty without spermatogenesis).
- ♀ → Virilism
  - 1ry amenorrhea
  - Hirsutism
  - dwarfism
  - Small breasts.
  - Acne.

Post Pubertal → Virilism
- 2ry Amenorrhea
- Hirsutism
- Breast Atrophy.

- Diagnosis: 17-OHP ↑↑ — Early morning Follicular phase → if equivocal → do either: ① ACTH Stim. test (if ↑↑ → CAH).
② 21 Hydroxylase enz level
③ Genotyping: CYP21A2

N.B if 17-OHP > 800 CAH diagnosis can

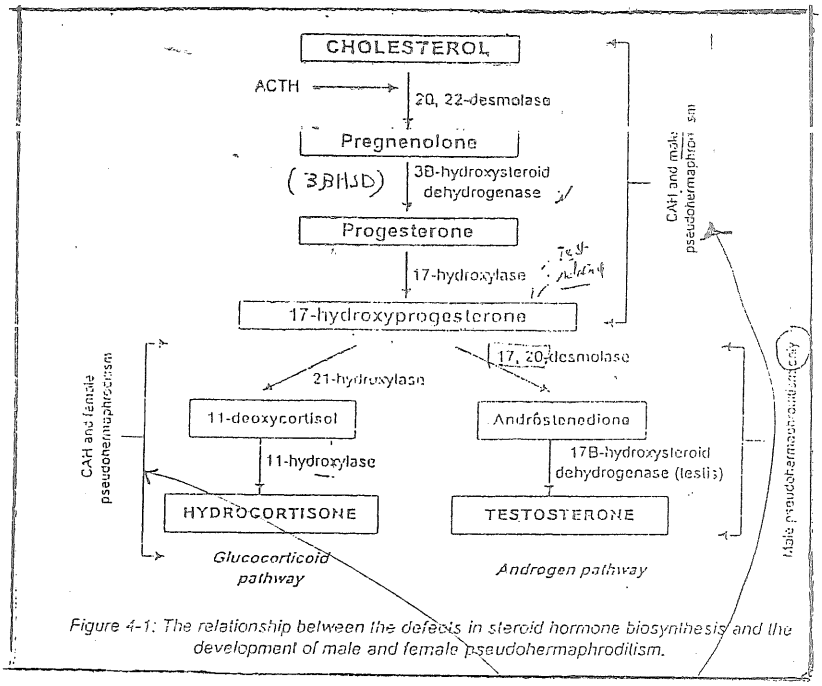


Figure 4-1: The relationship between the defects in steroid hormone biosynthesis and the development of male and female pseudohermaphroditism.

(B) . Adrenal Tms: (Virilizing Tm).

. Hirsutism d.t Adrenal Tm Ch By:

- . Age : 20 - 40 yrs.
- . onset : sudden
- . Course : Progressive
- . other Androgenic Manifs. e.g AGA, Acne

"cf" → . Hormonal findings:

- . DHEA-S > 7000 ng/ml
- . Total Test. > 200 ng/ml

(C) Adrenal Cushing → "see Pituitary" (adrenal Tm secretin
M. Cushing → ↓ SHBG → Hirsutism

- Pituit
- Adrenal
- Ectopic
- Iatrogenic

(NB)

• Cushing (Adrenal Hyperfunction) (or Excess Cortisol)

• ACTH dependant (↑ACTH)

1) Pituitary ACTH secreting Adenomas

2) Ectopic ACTH secreting Tm (Bronchial Carcinoma)

3) Iatrogenic: prolonged ACTH ↑. [For vitiligo]

• ACTH Independant (↓ACTH)

1) Adrenal Adenoma

2) Iatrogenic: Prolonged "Cs ↑."

- NB: the commonest cause of Cushing is: Cs ↑ but pure Cs use Cause Hypertrichosis not Hirsutism. So the presence of Cushing + Hirsutism (refer) to ↑ACTH & Adrenal Tm as both → ↑ Androgen + (Cs).

• Diagnosis of Cushing:

1) ACTH level
   - ↓ in adrenal Tm & Iatrogenic Cs
   - High in ACTH sec. Pituit. Tm
   - Very High in Ectopic ACTH Tm.

2) Dexamethasone Suppression Test:

   Dex. 2mg / 6hrs × 2 days.

   ↓

   Plasma Cortisol level assessment (Calss DIFFs & Tests ↓↓)

   - ↓ in Pituit Tm
   - ↑ in adrenal Tm & Ectopic Tm.

3) 24 hr Cortisol Level in (urine) is (the) Gold Standard for diagnosis.

4) Imaging
   - CT
   - MRI
   - U/S.

Evaluation of a Case of Hirsutism

A. History
B. Examination
C. Investigation.

A. History

1. **Race**: Females of < Arabic Gulf / [illegible] / [illegible] / middle East → have mild degree of Hirsutism which ± considered NL in these races.
2. **Age**:
   - Childhood: CAH.
   - Puberty (20-40 y): late onset CAH, Idiopathic (Most Common), PCOS.
   - Reproductive period: PCOS, Cushing, Tm.
3. **Onset**:
   - Rapid: → Tm. < Ovary / Supra renal gland.
   - gradual: → Idiopathic. (very slow & stable)
4. **Menstrual Hx**: Amenorrhoea or oligom.
5. **Drug Hx**: e.g. Androgen or Progest.
6. **Family Hx**:
   - +ve in Racial Hirsutism
   - CAH: → childhood dehyd. or Hx of precocious pub. in one brother.

B. Examination

General	local (Cut)	Genital	Pelvic
signs of virilization. " of Cushing.	stagin using (Ferriman-Gallwey Scoring System).	Ambiguous genitalia.	Enlarged ovaries, PCOS or Tm.

NB: Ferriman - Gallwey scoring system:

- a scoring system that asses the extent of hair growth in all androgen sensitive areas (9 areas)
- each area given score from: 0 (no excess terminal hair) to 4 (excessive terminal hair) frankly virile
- these areas are:
  - upper lip
  - chin
  - chest
  - upper back
  - lower back
  - upper abdomen
  - lower "
  - upper arms
  - thighs.

(This system → ...ld 11 areas / Modified 9 areas (forearms & legs were deleted).)

...: Modified → 19 areas.

- Def. of Hirsutism:
  - Score ≥ 2-3 in East Asian & Native American.
  - Score ≥ 6-8 in other population.

NB:
- Mild Hirsutism → Idiopathic, PCO, CAH.
- Severe " → Tms.

C. Inv

Lab
- Testosterone
- DHEA-S
- Prolactin
- Serum fasting insulin [illegible]
- 5-α reductase activity assessment
- Diagnosis of cushing
- " of CAH.

[illegible] حالة لازم تعمل تحاليل →

Rad.
- U/S
- CT
- MRI

↓ for Tms or PCOs

[illegible]

قاعدة
- mild & stable cases → no need for invs (usually idiopathic)
- Rapid onset or severe cases → do invs or ass. c̄ other androgenic manifs.

1. Testosterone → Free (preferred ??)

- Total: 70-90 ng/dl
بتقيسها

Vary during different phases of cycle ≈ 25%

↑↑ level (not) Correlated ē dis: activity as the dis caused by DHT.

level > 200 ng/dl → Tm

- Free T.: (done if Total T. is NL)

2. DHEA-S (↑ marker of supra-renal)

- level > 7000 ng/dl → adrenal Tm.

NB

- ↑ DHEA-S → Adrenal Canc.
- ↑ Testost. → adrenal (or) ovarian.
- ↑ Testost + NL. DHEAS → ovarian.
- ↑ Testost + ↑ DHEA-S → Adrenal.

Why ??

- Test. adrenal ovary > بيتكون
But
- DHEA-S بيتكون نسبة Adrenal.

3. Prolactin level (& inv. for ↑ causes)

4. Serum Fasting insulin: if Hirsutism ass. ē PCO, obesity, AN → Acanthosis Nigricans

5. 5-α reductase Enz. assessment: (نادر) [Controversy] مش بيعمل فايدة تقنية.

- Skin Biopsy
- Androsterone Glucoronide (بول)
- 3α. Adiol G.

↑↑ DHT Metabolite ←

6. Diagnosis of Cushing: ACTH level, ① dexamethasone suppr. test & 24hr Urinary Cortisol level.

7. Diagnosis of CAH:

@ Serum 17 OH Progest: (8bet 7-9 am) "قياس"

- Early morning
- Follicular phase

- ≥ 800 ng → CAH
- 200 - 800 ng → ACTH stim. test.
- < 7 nmol/L → NO CAH
- 7 - 15 n → ICAH → do ACTH if fear
- > 45 → CAH

فيه ثبات نفس (ACTH)

⑥ 21 hydroxylase enz. assessment.

↓ Sex Luteal PCO

المخطط (رسم شجرية)

Hirsutism

↓

- Baseline assesment of:
  - Free T
  - DHEA-S
  - Cortisol

↓

نعطي N للمريض

Dexamethasone 2 mg ld for 2 W.

↓

Repeat Androgen Assays

→ Suppressed androgens
  3 احتمالات
  - Idiopathic
  - CAH
  - ↑PRL.

  ↓ to diff.

  ACTH Stim. test
  - ↑ 17 OH Preg. / subNL Cortisol → CAH
  - NL 17 OH Preg. / ↑ Cortisol → Idiopathic or Hyperprolactinemia.

→ Non Suppressed Androgens
  يعني شي عم Androgen ??
  - PCOS
  - Tm.
  - Cushing (adrenal)

  to diff.

  ↓

  Cortisol leve

  ↓
  - ↓ in → PCOS, Tm. → Ovary, Pituit
  - Non ↓ → adrenal Cushing.

Treatment of Hirsutism

3 lines

بالتفصيل (See AV) ←
1. ↓↓ Androgen product. ⟶ Androgen inhibitors
2. Blocking Androgen Rs ⟶ "Antiandrogens"
3. Hair removal

---

1. ↓↓ Androgen product.

↓↓ Ovarian Androgen

[1] **Contraceptives**

(Est ⟨ ↓ FSH/LH → ↓ ov. and. ; ↑ SHBG → ↓ Free T ; ∴ 5α reduct.)

• Prog. ⟨ ∴ Rs ; ∴ 5α reduct. & – FSH, LH.

• Note: ∴ progest. has little effect on Hair follicles ∴ 5α reduct.

لازم تكون نوع من البروجسترون يكون "low androgenic"

↓ See AV

[2] Leuprolide: ∴ GnRH (Expensive).

↓↓ Adrenal Androgens (if CAH)

[1] Dexamethasone: 0.5 mg/d

[2] Predniscolone: 5 mg/d

(تعطى مساءً مره واحده)

↓↓ DHT product. from T

[1] Finasteride.

[2] Dutasteride.

Ketoconazole
∴ Both ovarian & Adrenal Androgen
How ??

(Used in Idiopathic Cases) ← 2. Blocking Androgen Receptors:

↓

Anti-androgens

1. Spironolactone (100 - 200 mg/d) → Cyclic (CPs لي) / Combined or

(* GIT upset)

2. Cyproterone Acetate < Androcure ; 10, 50 / Diane : 2 mg CA + 35 Mgm EE

3. Cimitidine

4. Flutamide

↓

S.E: Hepatotoxic   How to use (see AV)

• Other Medications: (Insulin Sensitizers or lowering Drugs..)

. Metformine (Cidophage) & Thiazolidinediones)

: Other Sensitizers rosiglitazone (2012)

. insulin sensitizing Agent → improve insulin sensitivity → ↓ ins. level → ↓ Test. level.

. Improve reproductive Funct- Not Hirsutism.

. dose: 850/d ↑ to 850×2

. S.E: Nusea, vomiting, diarrhoea.

• NB: • For Rx of Idiopathic Hirsutism: (With NL Androgen level).

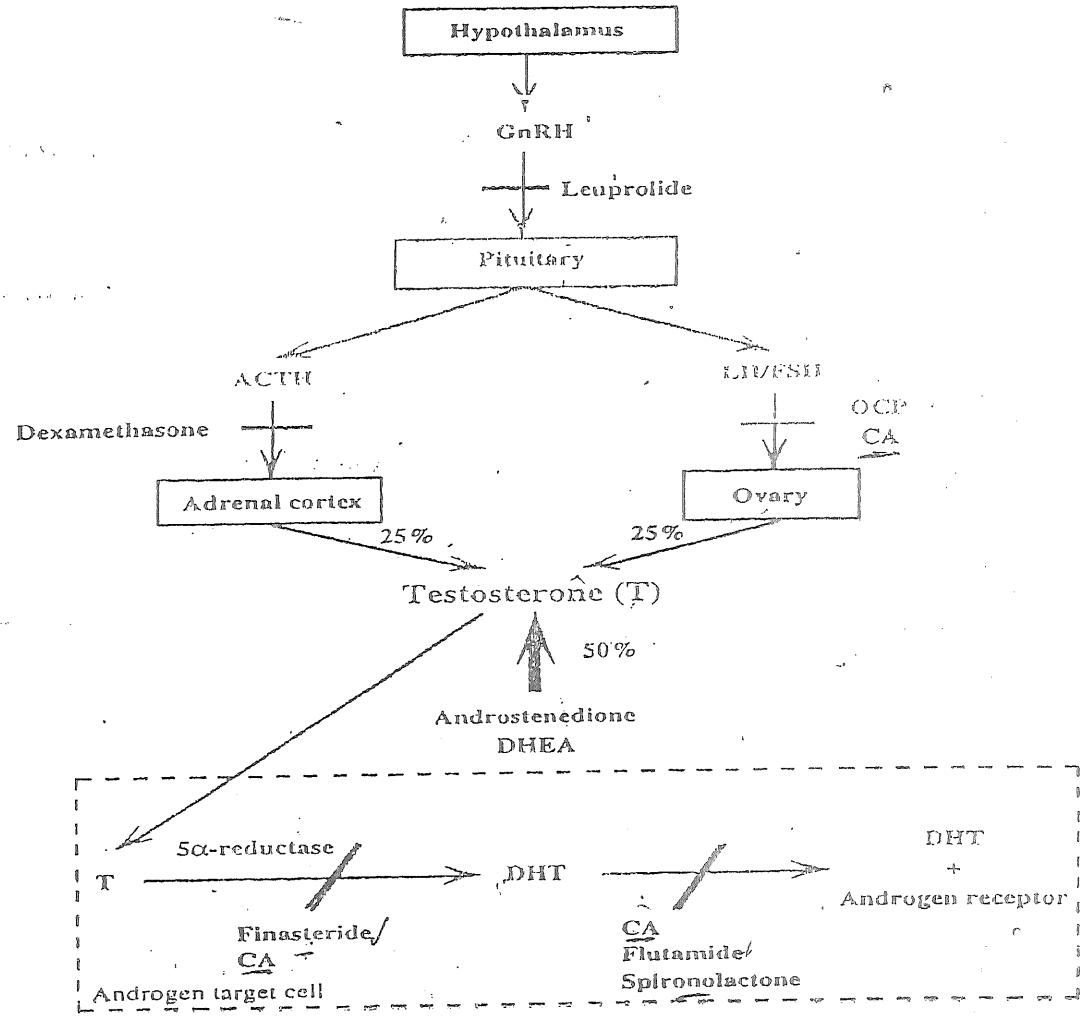
• Antiandrogens (- ARs).

• Finasteride & dutasteride.

: for ≥ 6-12 ms.

&

Should be avoided in pregnant (in ♂) or better Combined ē Contraceptives. (as Diane).



Site-specific blockage by different drugs used in the treatment of hyperandrogenism.

N.B.: uses of antiandrogens in dermatology:

- acne vulgaris
- hirsutism
- seborrhoea
- androgenetic alopecia
- hydradenitis suppurativa.
- Fox-Fordyce disease.

(SAHA)

- prostate carcinoma
- benign prostatic hypertrophy (BPH)
- breast cancer in men
- precocious puberty
- Criminal hypersexuality.
- Keloids & adhesions

- Antiandrogens have the possibility to feminize a male foetus & should only prescribed with adequate contraceptive cover.

SAHA. Synd : Seborrhoea, Acne — Hirsutism, AGA.
Frequently ass. é : PCOS, obesity, IR.

Hair removal

Physical methods of hair removal

- **Temporary hair removal** - Shaving, epilation (waxing, plucking, threading, sugaring, and using abrasives or mechanical devices), depilation, bleaching
- **Temporary hair reduction** - Eflornithine hydrochloride (VANIQA cream 13.9%)
- Permanent hair reduction- laser
- Permanent hair removal - Electrolysis

Bleaching

Bleaching makes the excessive hair less obvious by hydrogen peroxide

Depilatory creams :

Depilatory creams are generally based on thioglycolate (also used in perming solutions). A thick layer is applied for 15-30 minutes to the hairy area, then wiped off and the hair comes off with the cream. Depilatory creams can irritate and cause dermatitis.

Shaving

Shaving, if necessary twice daily, will prevent unsightly stubble. Shaving does not make the hair grow more thickly.

Waxing :

Waxing needs to be repeated every six weeks. The warm wax hardens on the skin and as it is stripped off, the hairs are pulled out with it from the roots.

Electric hair removers

These remove the hair by a combined cut and pull.

Electrolysis/thermolysis

Electrolysis or thermolysis may result in permanent hair loss but it takes time. A small probe is inserted along each hair, and a small electrical or heat discharge destroys the hair. A small area is treated every few weeks. It can be expensive if the area affected is extensive. Unskilled treatment may cause scarring.

Laser therapy

New long wavelength lasers and intense pulsed light are under investigation for the removal of body hair. Time will tell how effective these will be.

Eflornithine HCl 13.9% (Vanique): INHIBITS ornithine decarboxylase (enzyme essential foe hair growth). Hair growth inhibitor, not a depilatory. Twice daily cream

- reversed effect after 2mo.

Complications of physical methods of hair removal

Folliculitis is an unfortunate risk of plucking, shaving, and waxing. The treated hair follicles become inflamed, and painful pustules may develop.

Folliculitis may take weeks to settle. Hair removal has to be stopped, at least temporarily.

Andrews - Derozi, Emedicine

Hypertrichosis

Def Excessive growth of Hair (of any Type ← Lanugo, Vellous or Terminal) over Non Androgen dependant areas of SKin. Above the NL For ← Age, Sex, Race.

Types

- Localized
  - Cong / Hereditary
  - Acquired
- Generalized
  - Cong / Hereditary
  - Acquired

Localized Hypertrichosis:

Cong. / Hereditary

- Cong. Melanocytic Nevi
- Plexiform NF.
- Trichomegaly of Eye lashes. [ass ← Cong. HIV inf. & HH, SLE, Latanoprost, EGFR..., Malnut.]
  - (margin: ↑ length, Nof, No)
- Simple Nevoid Hyper-trichosis: Excess (terminal) Hair growth of Sacral area ass. ē → "Spina Bifida" [Faun tail sign].

Others.
Hair
(i) Collar sign ← Aplasia Cutis, ectopic neural tissue
(ii). Cubiti (Hairly elbow)
(iii). Ant. Cervical at laryngeal prominence.
(iv). Facial: Cornelia de lange Synd.

Acquired

- [Traum. — [illegible] (LSC)
- Vaccinat — [illegible]]
- [burns
- Scars.]
- [EBA.
- PCT.]
- Drugs (Topical) ← Tacrolimus, Cs., Minoxidil, Iodine.
- [Beckers Nevus.
- Scleroderma ← H. Syndrome, Melorheostosis]

NB. Facial Hypertrichosis ← familial, PCT, minoxidil, Cs.

- Hypertrichosis Singularis: one long wild hair.
- Systemic
  - JDM → infra patellar
  - Reflex Sympath. dysf.

Generalized Hypertrichosis

Cong. / Hereditary

1. Cong. Hypertrichosis Languinosa [CHL]
2. Universal Hypertrichosis
3. Prepubertal Hypertrichosis
4. Cong. Generalized ass e

Syndromes:

- Mid facial vasc. lesions
- Broad thumb
- Straight nose
- High arched palate
- MR
- Short st.

→ • Rubenstein Taybi
- Leprechaunism
- Hurler
- Stiff skin synd.
- Cornelia De Langes synd.
- Ambrass Synd. (Werewolf Synd) ذئب
- Lipodystrophic synds.
- tw

Acquired

- Mg associated → Acquired HL (AHL)
- Non Mg ass.
  - Hypo & Hyperthyroidism
  - Acrodynia
  - EBA
  - DM (Dermatomyositis)
  - Encephalitis.

Iatrogenic

- Immunosuppressive:
  - Cyclosporine
  - Minoxidil
  - Hydantoin
- Others:
  - Streptomycin
  - Cs ✓
  - Psoralens ✓ (PUVA)
  - Diazoxide
  - Penicillamine
  - Acetazolamide.

- NB Hypertrichosis Languinosa (HL)
  - Congenital (CHL)
  - Acquired (AHL)

سؤال امتحان

NB Universal Hyperbrichosis: variant of CHL but hair is more thick & longer, Mainly at face, ears & shoulder
. Persist throughout life.

More:
- longer
- Thicker
- Persistent

CHL

. Very rare disorder; there is failure of replacement of Lanugo Hair by Vellous

. CHL ← Dog or Monkey Face"
- Generalized Hypertrichosis (Except palm, soles, Genitals)
- blond - gray unpigm. & Fine Hair.
- ↑ growth from birth to 2 ys → ↓ Age of maturation of puberty

. May be ass. :
- glaucoma & Pleiotrophia
- Ear anomalies
- dental anomalies
- pyloric stenosis
- GR
- MR

AHL = Malignancy

. Lanugo hair may develop :
1. all over the body
2. localized to the face → "Simian appearance"
3. AGA areas.

. May be ass. with :
1. Mg = this a para Neoplastic dis. Commonly ass. with Cancer of lung, Colon or Breast.
2. other Paraneoplastic diseases :
   - AN [Acanthosis Nigricans]
   - Leser Trelat sign.
   - Acq. Ichthyosis
3. Tongue disorders e.g burning pain.

. NB : **Prepubertal Hypertrichosis**
- affect infants & children of < Mediterranean / Asian descent.
- Generalized & more evident at < Face / Temples / Preauricular > back (inverted Fire tree), Bushy eye brows, low ant. Hair lines.

• Hair Shaf Defects

Classification

I. Associated ↑↑ fragility.

II. Not " " " "

III. Other defects.

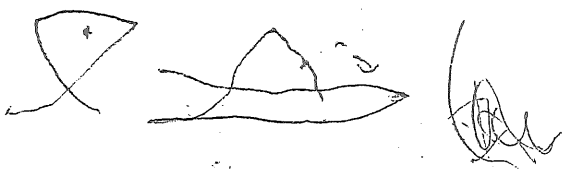
I. Defects Associated with ↑↑ Fragility:

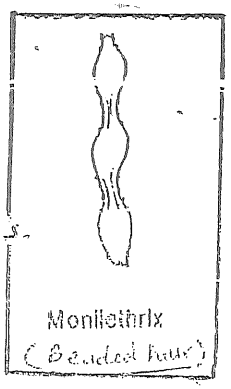
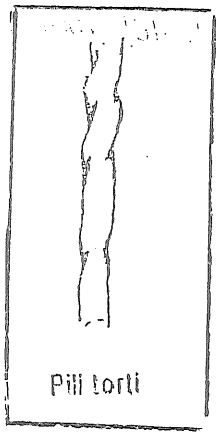
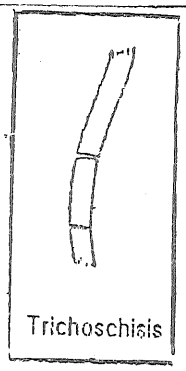
Hereditary

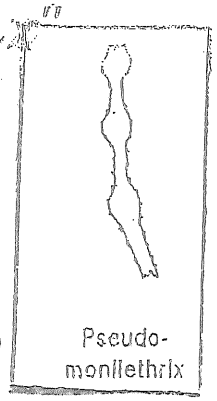
- Monilethrix
- pili torti
- Trichorrhexis Invaginata
- Trichoschisis
- T. Nodosa

Acquired

- Trichorrhexis nodosa
- Trichoptirosis
- Trichomalacia
- Pohl pinkus Constrictions
- Bubble Hair.

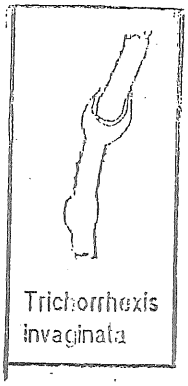


Dis.	pic.	Etiology.
1.) Monilethrix Beaded Hair = Elliptical Nodes Constrictions at = Imm intervals).	 Monilethrix (Beaded hair)	• AD disorder d/t defect ē K1 & K6 • ± ass. ē ↳ KP (K6 defect) ✓ [. Cataract ✓ [. MR [. Menkes . Kinky Synd. • DD → Pseudomonilethrix [Flattening or indentation ē protruding Edges] Monileth → • Ht ← KP → Ectodermal Griseof. → for growth ↑↑ ↓ Trauma
Pili torti (twisted Hair)	 Pili torti	• Etiology ± : Hereditary → Isolated / ass. ē Synd. ✓ Bazex Synd. ✓ Hypohidrotic Ectodermal Dysplasia [. Menkes Synd [. BIDS. Acquired ✓ Retinoids at Edges of Cicat. Alopecia. Dysplasia → Hypohidrosis / Hypotrichosis / Abnormalities.
Trichoschisis Transverse Fractures)	 Trichoschisis	• d/t Genetic defect → ↓ sulfur Content of Hair & defective DNA repair Mechanism → Photosensitivity • Ass. with "Tricho thiodystrophy" Synd: (3 synds): • BIDS . Brittle hair (Trichosch.) . Intellectual impairment . Decreased Fertility . short Stature. • IBIDS → as BIDS + Ichthyosis • PIBIDS → as IBIDS + photosensitivity.


Pseudo-monilethrix

(4) Trichorrhexis Invaginata

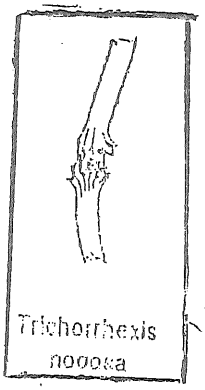
(Bamboo = Ball & socket = Intussusception)



- one part of shaft is invaginated in the other (Intussusception).
- T. Invaginata = Netherton Synd
  - Ichthyosis — Erythrodermic Ichthyosis linearis Circumflexa
  - AD
  - FTT
  - T. invaginata
- Eczematic.

(5) Trichorrhexis Nodosa

(Broom stick) مكنسة زرقاء



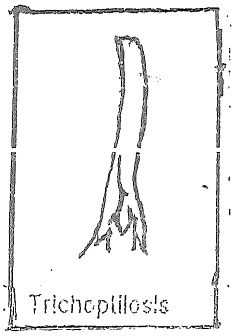
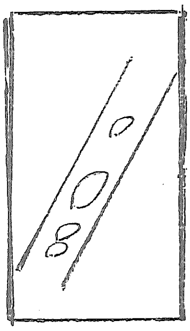
مقشتين متقابلين

(i) - Netherton
(ii) Argininosuccinic A.
(iii) Strongylenosis.

- (Tissue) Fracture of Cuticle & Cortex → their fragments "splay out" like Ends of 2 brushes pushed into one another.
- Commonest Hair shaft defect.
- CIP: grayish white Nodes & fracture of Hair.
- Etiology (A) Cong < argininosuccinic aciduria / MR / ± Netherton.

(B) Acq.
- Proximal → بعد استعمال كيماويات وأفران ساخنة
- Distal → repeated Cumulative Trauma
- Circumscribed → at beard & Moustach

(1) NB Other ass. Hair defects:
- Pili torti
- Ribbon like: Hair shaft Flattened & folded over its self.
- Alternating white & dark bands.
- Damaged cuticle.

⑥ Tricho-ptilosis (longitudinal splitted ends) الأطراف المشققة		. Caused by recurrent trauma . ± ass ē < Long Hair / Trich. Nodosa . Ht : → # Trauma / → Cut Splitted Ends
⑦ Tricho-malacia (disintegrat. at suprabulbar area)		. Et = "Trichotillomania" ([illegible]) . Hair is broken at variable lengths → patchy alopecia ē some follicles plugged & contains deformed swollen hairs [dark bodies].
⑧ Pohl-Pinkus constricts. (اختناقات)	اختناقات بتحصل في فترة توقف أثناء العلاج الكيماوي	. = "Anagen Effluvium" . Similar to "Beau's lines" of Nails.
⑨ Bubble Hair بيكون فيه فقاعات هواء.		نتيجة استعمال السيشوار

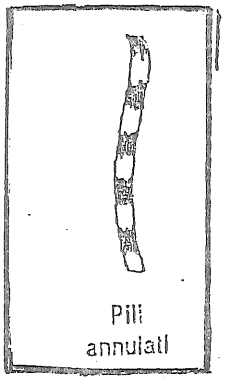
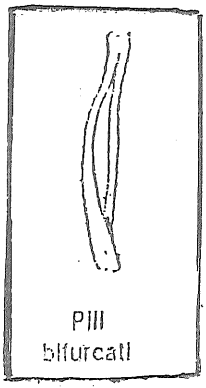
Hair ↓ < Sulfur → PIBIDS / Copper → Minkes Kinky

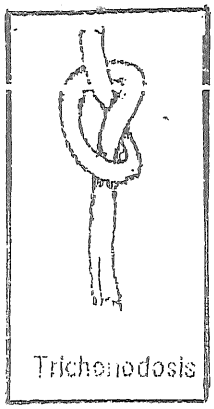
- Menkes' Kinky Hair Synd. (XLR)
  - d.t ↓ Copper content of Hair & Serum d.t ↓ intest. abs.
  - C/P:
    1. Hair shaft abnormalities (Monilethrix, P. torti, T. Nodosa)
    2. Vascular abnormalities
    3. GR / MR
    4. Others: Pudgy Face, Ptosis, Neurological Manifs.

II. Defects Not Associated ē ↑↑ Fragility (10).

- Pili < Annulati / Multigemini / Bifurcati

- Trichonodosis
- Trichostasis Spinulosa
- uncombable Hair Synd ✓
- Woolly Hair Nevus.
- Straight hair N.
- Acq. Progressive Kinking.
- Loose Anagen Hair Synd.

Pili Annulati (Ringed Hair) خطوط أساسية فاتحة.	 Pili annulati	. air Filled Cavities: الأساس فيها فقاعية . Hair growth is NL & usually improves ē Age. . DD: Pili pseudoannulati خطوط أساسية فاتحة وأساس متباعد. . NL Hair Variant
Pili Bifurcati (متشعب أساس ويحتوي أساس اثنان) [Separate & Fused rami]	 Pili bifurcati	. dt Formation of 2 different Sized shafts [ē Separate Cuticles] by Single Matrix.
Pili-Multigimini (Tufted)		. Bifurcated or Multiple divided hair Matrices & papillae → Multiple shafts originating From one Follicle . Each shaft has its own IRS but the ORS is Common For all.

Dis.	Pic	Discussion.
• Trichonodosis (Knottings) عقدة	 Trichonodosis	. In Blacks in response to Trauma.
. Trichostasis Spinulosa معوقة جريان (البثور السوداء) (الانف)		By Keratin. . Follicular Plugging + retained bundles of Vellous hair → Black head like at Nose, forehead & Cheeks. . Hx: • Plugging → • Retin A • Biore Plaster (بلاستر البثور) • Hair → • Waxing • Laser.

Uncombable Hair Synd (Spun glass Hair) = Pili Trianguli Et Canaliculi

- Hair is stiff & difficult to comb.

"Age" → Mic. exam ≤ Longitudinal groove
Flattening (ribbon like)
hair is △ in cross section

- Def.
- Er.
- Clp.
- Mic.

- AET: unknown but may be d.t abnormal Keratinization of IRS

- Clp: at first few years after birth: Hair is:
  - dry, blond, shiny
  - stands straight out from scalp
  - Can't be combed.

- Types ± ≤ AD / AR / Sporadic.

- may be noted in NL Hair but affect < 50% of scalp Hair; So if ≥ 50% affected → "Uncombable Hair Synd"

- ## → ① Usually no ## but one case Responded to Biotine 0.3 mg x3/d. (Shelly 85)

② May improve Spontaneous.

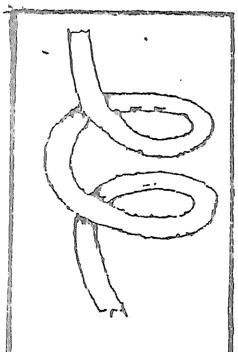
• Woolly Hair (W.H) (شعر مجعد) = "Curled Hair"

- Def: Presence of Woolly or unruly (Negroid Like Hair) or Coiled Hair, involve the whole scalp (Woolly Hair) or Circumscribed area of scalp (Woolly Hair Nevus) on the scalp of non Black Persons (non Negros).

- ±: AD, AR

Clinically:

- usually start at birth & ↑↑ in severity in childhood.
- Woolly Hair Ch. BY (شكل الصوف أو يكون شكل شعر النيجرو ومنبعج مع بعضه)
  - ↓ diameter
  - ↓ growth (may not grow > 12 cm).
  - unite into Tight locks → difficult brushing
  - ↓ color (may: lighter).


Woolly hair

(unite into Tight locks)

لو شفت Woolly Hair

افحص

- PPK
- Sole
- Heart

Associations:

① PPK + Cardiomyopathy

- diffuse non epidermolytic PPK. (d.t Plakoglobin Mutation) → Naxos dis
- Striate Epidermolytic PPK (d.t desmoplakin Mutation) → Carvajal synd.

② Noonan Synd

③ K.P

④ Icthyosis

⑤ ulerythema ophryogenes.

. Straight Hair Nevus :

- Straight Patch in Kinky hair scalp

NB

- Kinked (Curled) Hair [of negro] if develop on scalp of Straight Hair [eg whites] → Woolly Hair Nevus.
- Straight Hair [eg whites] if develop on scalp of Curled (Kinked) Hair (eg Blacks) → Straight Hair Nevus.

Acquired Progressive Kinking (Whisker Hair)

- Acq. Progressive Kinking & Twisting of Hair at regular intervals · usually affect men ≃ 20 y.
- Site: Start at frontoparietal or vertex regions & then progress to both parietal & Temporal areas
- Hair: Frizzy, Curly & Lusterless
- AET:
  - ① AGA : Represent precursor of AGA (Patients usually have Hx of AGA)
  - ② Retinoids.
  - ③ HIV.

Loose Anagen Hair Synd

- Cuticle of HRS → Ruffled = Rumpled sock

Loose anagen hair syndrome

This condition features anagen hairs that are loosely attached and easily pulled from the scalp. Most cases are female children. Inheritance is autosomal dominant. The patients typically have slightly curly hair, which is of uneven length, and patchy in quality. Variants include those with stiff, uncombable hair and those in whom shedding is the primary complaint. The children may present with patchy alopecia which is due to hair pulling. Trichograms show 100% anagen hairs. Hair is usually easily and painlessly plucked with the hair pull test. The hair becomes normal with age. Minoxidil 5% topically may be of value.

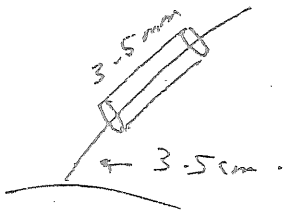
III. Other Hair shaft Dazects.

1. Hair Casts (Pseudonits)
2. Trichoclasis (Green stick fracture)
3. Circle Hairs (ingrowing Hair): Pseudofolliculitis barbae.

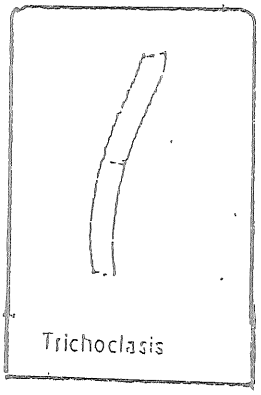
---

(1) Hair Casts: (Pseudonits):

- represent remnant of inner root sheath. (IRS)
- They are white-keratinous sleeves, 3-5mm length, beyond 3-5 cm from the scalp & can (unlike nits) slides along the shaft
- occurs in female 2ry to trac̈t.



- ACT:
  1. in ass. c: Tract hair styles, Hair sprays used, Persistent resistent clamdrop
  2. May be unusuall manifestat of "psoriasis".
- Woods light: Blue-Yellow Fluroscence.
- III → Retin A.



(2) Trichoclasis (Green stick Fracture): Transverse Fracture of Shaft that partally splinted by intact cuticle.

(3) Ingrowing Hair: see Pseudofolliculitis barbae.

NB : Hair shaft Examination :

- Puting piece of double shck Tape on Mic. slide & aligning 5 cm segments of Hair in parellel on it

↓ then examine BY

- dissecting Mic
- Polarized light
- Gold Coating & scanning EM

Hair Color.

- There are 3 types of Melanosomes:
  - Brown & dark Hair → Eumelanin
  - Blond Hair → Pheomelanin
  - Red Hair → Pheomelanin & Erythromelanin.

- Hair color depends upon the amount & distribution of Melanin in hair shaft (cortex):
  - Brown & Black → many melanized eumelanosomes.
  - Red hair → pheomelanosomes.
  - Blond → fewer or incompletely melonized pheomelanosomes.
  - Gray → Few MCs or incompletely melanized melanosomes.
  - Snow White: Absent MCs
  - Albinos: MCs present but Enz Tyrosinase enz.

- Sites of Melanocytes in Hair follicle:

① Melanotic MCs: Interspersed bet. cells of the Matrix Capping the Dermal papillae.

② Amelanotic MCs: in the outer Root sheath they form melanin only after skin injury (after Dermabrasion). or Br UVL (perifollicular pigm. in Vitiligo)

(تابع)

- Whitening = Graying of Hair
  - Diffuse: Canities
  - Localized: Poliosis.

Canities.

Causes

- Physiological
  - Aging:
    1. ↓ Tyrosinase Activity.
    2. Defective MCs migration.
    3. ROS Mediated DNA damage of MCs.

- Pathological [premature graying of Hair] "شائع"
  - def. Graying occurs Before:
    - Age of 30 in blacks (Asians)
    - Age of 20 in whites
  - Causes:
    1. usually Familial.
    2. Emotional stress.
    3. pernicious anemia
    4. thyroiditis (Hypo or Hyper).
    5. Alopecia Areata (diffuse)
    6. Synds:
       - premature Aging e.g Werners
       - Rothmund Thomson

- ttt of premature Graying ??
  1. PABA: whiten the Gray hair in 80% of Patients after 1 month of ttt (300 mg/d)
  2. Hair dyes

Poliosis (& premature Graying نفس الحاجة) "شائع"

def. Gray or white hairs occur in Circumscribed Area or patches (Vitiligo, Piebaldism, Waardenburg Synd., AA, NF. & TS).
(Localized)

Acne TTT: Suppression of inflammation

- Inhibitors of chemotaxis: tetracyclines&erythromycin
- ↓↓ ROS: tetracyclines, erythromycin & azelaic acid
- -- Severe/late inflammation: Isotretinoin&tetracyclines

- **Dapsone:** The sulfone agent, dapsone 5% gel, is available as a twice-daily agent for the therapy of AV. The mechanism of action is generally thought to work as an antiinflammatory agent. The benefit in women seems to exceed the benefit in male and adolescent patients.
- **Nicotinamide:** Nicotinamide, also known as niacinamide or nicotinic acid amide, is the water-soluble, active form of vitamin B3. It has been increasingly studied for many different indications in the field of dermatology but more research is needed to clarify its value.

- Nicotinamide is naturally present in small quantities in yeast, lean meats, fish, nuts and legumes. It is also often added to cereals and other foods. Oral nicotinamide is available as 20-30 mg in multivitamin combinations, and on its own as inexpensive 500-mg tablets. It has also been incorportated in many topical agents including sunscreens and cosmetic agents.

Nicotinamide, available in topical cream, gel and oral forms (e.g. trade name Nicomide), has been shown to be effective in clearing acne. In a controlled clinical trial, 4% nicotinamide gel was found to be as effective as the topical antibiotic 1% clindamycin gel in the treatment of acne vulgaris in 76 patients with moderate acne. The study concluded that the anti-inflammatory properties of nicotinamide may have contributed towards its success in acne.

Nicotinamide also reduces facial sebum production. Sebum is responsible for facial shine and contributes to noninflamed comedones and inflammatory acne lesions. Results of a well-controlled clinical trial in Caucasian and Japanese women have shown that application of 2% nicotinamide moisturiser to the face for 4-6 weeks reduces sebum production with significant differences in facial shine and oiliness.

Nicotinamide gel is marketed as an over-the-counter treatment for acne in Canada, Australia, NZ, UK, USA and Ireland. If twice daily application causes excessive drying of the skin, one may reduce to one application a day, or every other day.

Nicotinamide is not recommended for acne in pregnant and nursing women.

Rosacea

I-Topicals

J Am Acad Dermatol
Volume 72, Number 5

Table : Topical medications shown to be beneficial in the treatment of rosacea*

Medication name	Level of evidence	Mechanism of action
Treatments approved by the FDA		
Sodium sulfacetamide	IA	Unknown, but likely antiinflammatory[17,20]
Metronidazole	IA	Decreased ROS generation and inactivates existing ROS production[18]
Azelaic acid	IA	Decreased expression of KLK5 and cathelicidin[19]
Alfa-adrenergic agonists	IB	Vasoconstriction of smooth muscles surrounding vessels of the superficial and deep dermal plexuses[15]
Treatments not approved by the FDA		
Retinoids	IIB	Connective tissue remodeling[21,22] and TLR2 downregulation[23]
Calcineurin inhibitors	IIB	Antiinflammatory[24,25]
Benzoyl peroxide	IB	Unknown
Permethrin	IB	Antiparasitic properties treat cutaneous demodicidosis[13,26]
Ivermectin[†]	IB	Antiparasitic properties treat cutaneous demodicidosis[26]

Brimonidine : The first and only FDA-approved topical treatment for the topical treatment of persistent facial erythema (redness) of rosacea in adults 18 years of age or older.

- **Mechanism:** selective alpha-2 adrenergic agonist.
- **Application:** Applied once daily, brimonidine works quickly to reduce the redness of rosacea and the beneficial effects last up to 12 hours.
- **Side effects:** In the long-term study, where patients used brimonidine topical gel for up to 12 months, the most common adverse events included rebound flushing (10%) and erythema (8%), rosacea (5%), nasopharyngitis (5%), skin burning sensation (4%), increased intraocular pressure (4%), and headache (4%).

TABLE 2: TOPICALS USED OFF-LABEL FOR ROSACEA

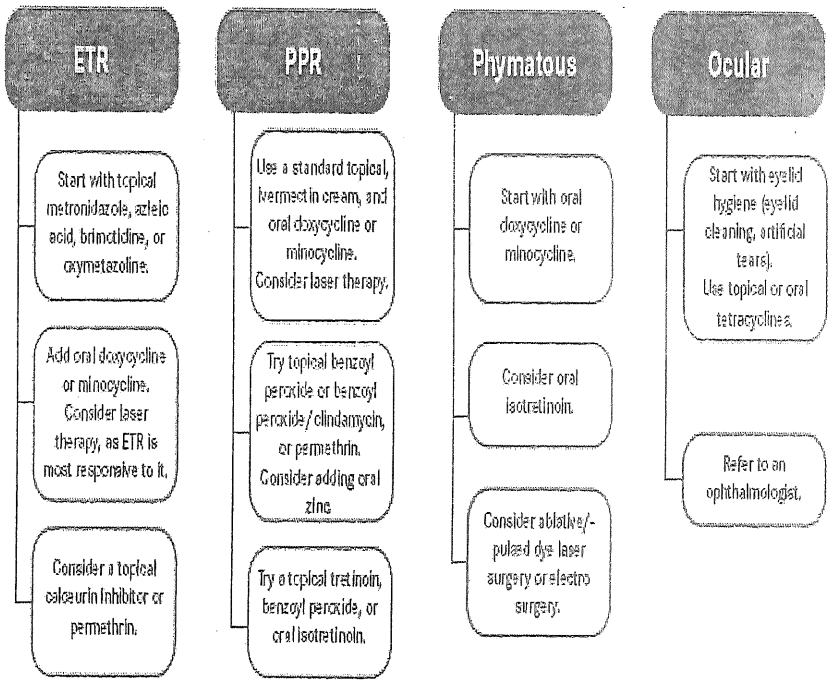
Product	Target Symptom	Comments
Calcineurin inhibitors (tacrolimus, pimecrolimus)	ETR or PPR	Usually leads to good response; specifically for inflammatory lesions; may cause burning, itching, or stinging
Permethrin	ETR or PPR	May be as effective as metronidazole 0.75% gel for erythema and papules
Benzoyl peroxide	ETR or PPR	Often effective, but may cause itching, burning, and bleaching of hair and clothing

ETR = erythematotelangiectatic rosacea; PPR = papulopustular rosacea.
Adapted from references 4, 12, and 13.

II-systemic TTT:

- Tetracyclines
- Azithromycin
- Isotretinoin (low dose 10mg daily)
- Inderal
- Metronidazole

FIGURE: TYPICAL PRESCRIBING AND TREATMENT PATTERNS FOR ROSACEA

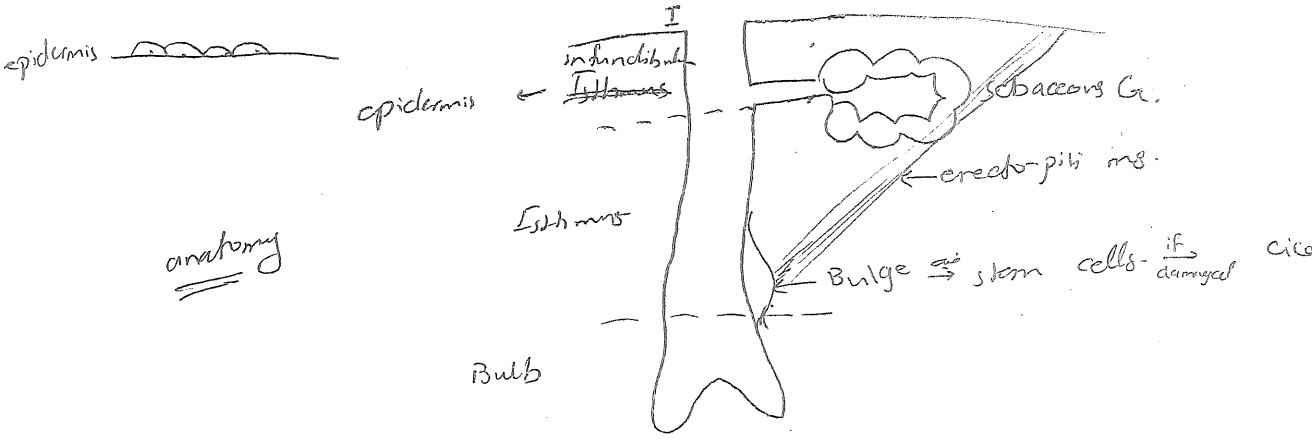


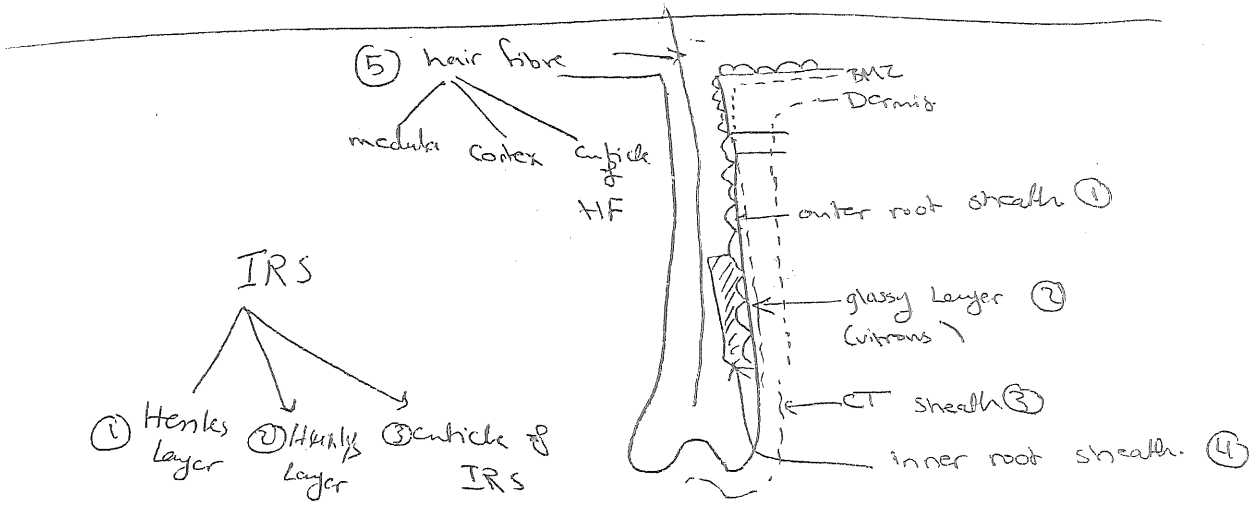
ETR = erythematotelangiectatic; PPR = papulopustular rosacea.
Adapted from Reference 4.

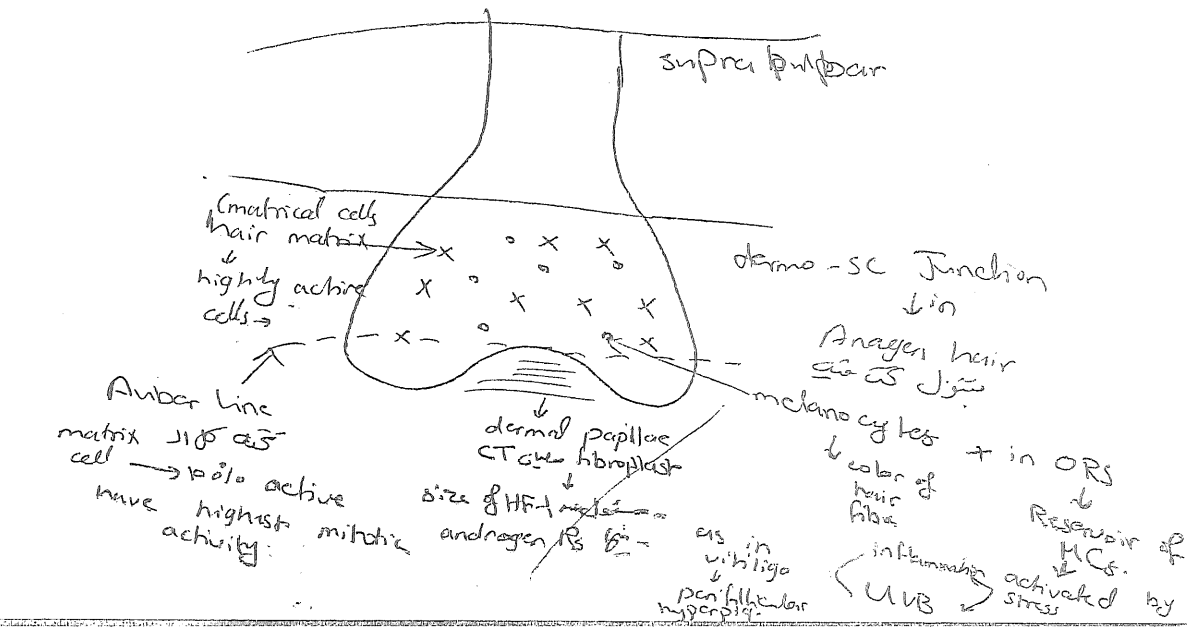
* topical
Nicotinamide = Niacin = Vit B_3 (Ebaglo)
↳ anti inflammatory

Hair

structure







3

Alopecia

cicatricial a. (scarring) — دكتور أحمد

non cicatricial

(A) congenital → Atrichea, hypotrichia → cong. triangular alopecia → H trial of minoxidil بيعمل نمو بسيط

(B) Acquired → localized → AA, Artifectual, AGA
→ Diffuse → TE

1ry
- HF مضروب
↓ Bulge
1ry target of inf. is → HF

2ry
- congenital → aplasia cutis congenita, Naevus sebaceous
- traumatic → caustic, surgery, thermal
- infections → TB, kerion, leishmania, HZ
- Neoplastic → BCC, SCC
- others → morphea, sarcoidosis, Necrobiotic lipoidica

ACC → erosion in any part of skin (Bart's syndrome)

(en cup de sabre) morphea → كأنه مضروب بالسيف

1ry: A → lymphocytic predi.

(1) → DLE → atrophy, scarring, telangiectasia, dyspigmentation

(2) → Lichen planus → LPP → scaly plaques; FFA; GLS (graham little synd.)

(End stage of any type of cic Alo)

(3) central centrifugal cicatricial alopecia (CCCA)

(4) pseudo pelade of Brocq → porcelain white, atrophic, depressed scar with no sign of inflam; middle aged female.

follicular lichen planus → cicatricial a. of scalp; Non " " of pubis, axilla.

(5) follicular mucinosa: excess mucin deposition in seb. g. & h.F.

C → erythematous plaques, بقع ساد... site → face, neck, scalp?

D/D of indurated erythematous plaques?
→ A-mucinosa
→ DLE
- tumid
- pseudolymph.
- lymphoma
- Jessner lymphocytic inf.
- granuloma faciale.

B → Neutrophilic type

C → Mixed type

D → other.

*cycle → arrest & anagen → arrest () stage III - IV → acute
↳ inflammation → Dystrophic anagen
↳ Disturbance at telogen → chronic

*Theory → immune previlage theory → Ag hidden in area → immune syst- not identify it ↳ as protein of eye lens ↳ sperm.

normaly → HF have no MHC I & II so in AA → MHC I, II (Ag) appears on HF → AB - attack

Cp
→ Any age
strong → AA. Atrechia
↳ sudden onset in completely normal skin.

exclamation mark hair
↓
- Active AA
spreading AA

clinical varities ① classical type (Localized)
② ophiasis → hair line → bad prognosis
sisaipho ← aff. affecting
⑥ Totalis
⑦ universalis

⑤ AA diffusa = Incognito → rapid diff. hair loss seems TE
⑥ Linear AA.
classification → Iked classification. CAPA
① → common.
② Atopic
③ Pre hypertensive
④ Auto immune

Q1 AA ∈ bad prognosis ?
① → onset
↓
Young < 5 yrs.
→ coarse
↓
progressive
→ Duration
↓
> 5 yrs
→ Side
↓
= extrascalp
→ type
- erythtable patchy
- ophiasis
- totalis
- universalis
→ association
- pitting
- AD

Previelage

* Androgenetic A *

* androgen → testes, ovary / → adrenal — ع بيشتغل → seb. g. / HF
* androgen Rs → more in frontal ~~parietal~~ vertex / → less in occipetal, parietal

androgen → Beard, mostache — vellus → terminal — Hirsutism
→ scalp — terminal → vellus — AGA

(1) terminal: vellus — N: 8:1 / AGA 4:1

(2) Shedding: ↑ telogen / anagen duration → ↑ shedding / → ↓

* Exogen →

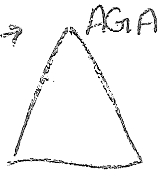
(3) * Kenogen HF → telogen وقت → anagen
(empty HF) — ما طالع

AGA → in female = FPHL → central thinning → diffuse / Ludwig / increase (olsen) الأقاني بحص
↳ nevere affecting hair line.
exception Hamilton → exception.

Dermoscope → hair shaft diversity
→ vellus hair ↑
→ Peripilar casts = degenerated scales

* investigation → in FPHL ē → Hirsutism / Acne / obesity / insulin Rs.
- TE → iron / thyroid
→ Trichoscopy.
→ trichogram → T:A 1:5 / V:T 1:4

→ AGA in male



C.H.D
Lobule of ear
Diagonal ear lobe of neck

no minoxidil ← لو حاملا

Tt → Minoxidil
→ finasteride, dutasteride
→ low level laser light therapy
→ PRP
→ Bimatoprost (Lumigan)

A+ opener → ~~explain~~ → ↑ K ch
بحضن ↓ K

- VD
- PGF like mechanism. → ↑ hair growth
↓ Lumigan — بيشتغل عالي
↓ EGF
→ mitosis of KCs.

*/ ⇒ ① hair follicle < A / T → mini anagen → miniaturization. (7)

→ infiltration → lymphocyte (swarm of Bees)
- peri Bulbar
- track of inf. cells.

CD4 : CD8
4 : 1

leukocyte
- Granular
  → Neutro
  → eosinophil
  → Basophil
- non granular
  → lymphocyte
    - T
      - helper → CD4
      - cytotoxic → CD8
      → T reg. ↓ FoxP 45
    - B → plasma cell → Igs.
  → monocyte (Bl.) → macrophage (tissue.)

*CD8 → عسكري

→ any tumour cell or virus enter cell

*CD4 ⇒ ظابط

Ag → comes on cell (Anti presenting cell)
بكسر الاكتشاف
CD4 → ظابط → cytokines.

*TTT ⇒ ① Reassurance if localized, limited duration → 80% (بتخف)
② تقدير خطورته،

- < 10 yrs
  ① Minoxidil
  ② ± CS
  ± Anthralin
- > 10 yrs.
  - < 50% → ILCS ±
  - > 50% topical immuno therapy.

CS → syst. → mini pulse → 2-10 mg Dexamethasone مرتين / w.
↓ ½ amp. epidrone مرتين / w.
to prevent progress
stable لمرض
حسب حالته

من يحصل للشعر في أسبوعين
لو ال IL زي طبقة ← تكسيش ← او قف

→ topical → super potent → Dermovate

*IL → kenacort 1ml → 40mg triamcinolone
trunk 1:4 → 10mg/ml
1:8 → 5mg/ml
1:16 2.5mg/ml ← face, wide area
لمدة ٤-٦ / اسبوع

* tricho tillomania *

girl → 5-12/18

* pathogenesis *

anxiety

* pathology
- trichomalacia → fragmentation of HS
- collagen cleft
- necrotic ks
- RBCs extravasation.

trichoscope → Black dot
- variable lenghth
- coiled hair

* trichophagia → tricho bezoar
anychophagia.

window test → JE10 → shaving for 2cm → growth نمو الشعر

N- Acetyl cystein → neuromediators effect
1200 - 1400 /day.